

Explatiation.

Abciximab (ReoPro) is a chimeric monoclonal antibody that is a glycoprotein Ilb/Illa receptor antagonist. It inhibits platelet aggregation and is mainly used during and after coronary artery procedures such as angioplasty.

It is advised that baseline prothrombin time, activated clotting time, activated partial thromboplastin time, platelet count, haemoglobin and haematocrit are all measured at baseline before use. Haemoglobin and haematocrit should be measured again 12 and 24 hours after commencing treatment and the platelet count 2-4 hours and 24 hours after starting treatment.

The EPIC trial showed that the use of abciximab reduced the risk of death, myocardial infarction, repeat angioplasty, bypass surgery and balloon pump insertion when used for high-risk patients undergoing angioplasty. www.ncbi.nlm.nih.govi

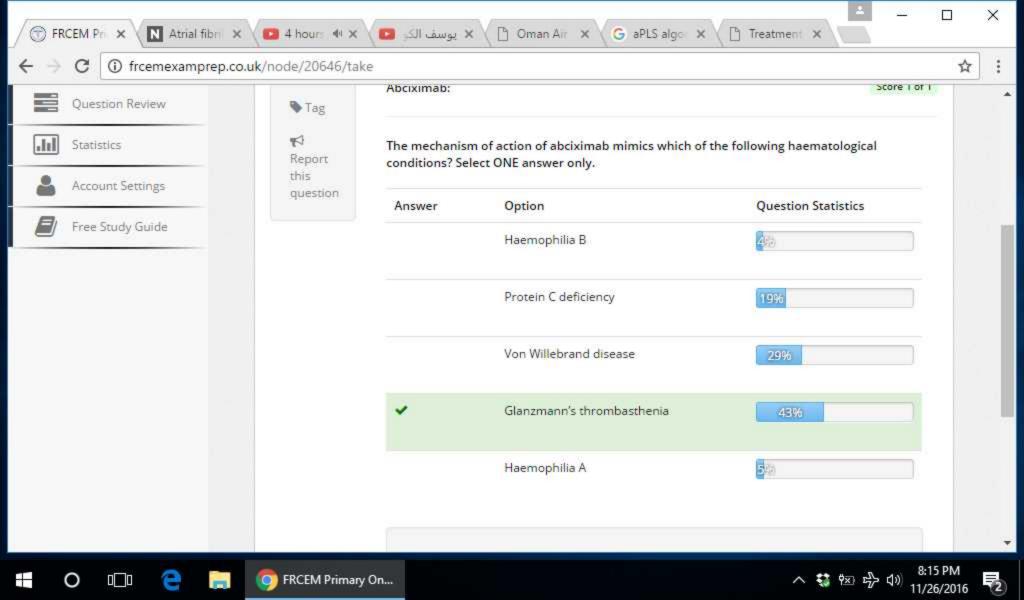
Abciximab is safe to use in the presence of chronic renal insufficiency.

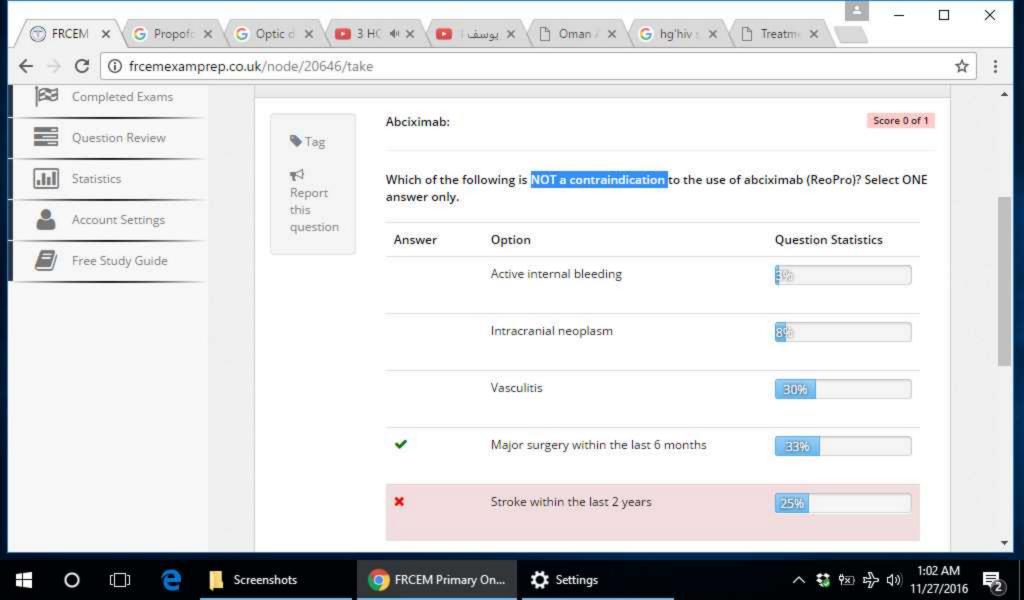
The following are contraindications to the use of abciximab:

- Active internal bleeding
- Major surgery, intracranial surgery or trauma within the last 2 months
- Stroke within the last 2 years
- Intracranial neoplasm
- Arteriovenous malformation or aneurysm
- Haemorrhagic diathesis
- Vasculitis
- · Hypertensive retinopathy

The side effects of abciximab include:

- · Nausea and vomiting
- Bleeding manifestations
- Bradycardia
- Chest pain
- · Back pain
- · Puncture site pain
- Thrombocytopenia
- · Cardiac tamponade (rare)
- Adult respiratory distress (rare)





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1:19 AM

⊕ 76% 
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# **Explanation:**

Allopurinol should not be commenced during an acute attack of gout as it can both prolong the attack and precipitate a further acute attack. In patients already established on allopurinol it should, however, be continued and the acute attack treated as normal with NSAIDs or colchicine as appropriate.

The first-line treatment for acute attacks of gout is non-steroidal anti-inflammatory drugs (NSAIDs), such as naproxen. Colchicine can be used in circumstances where there is a contraindication to the use of NSAIDs, such as in patients with hypertension and those with a history of peptic ulcer disease. This patient has no reason to avoid NSAIDs and therefore naproxen would remain the drug of choice from the list of options above.

When the acute attack has settled it would be reasonable to titrate up the dose of the allopurinol, aiming for plasma urate levels of less than 6 md/dl (< 360 µmol/l).

Febuxostat (Uloric) is an alternative to allopurinol used in the management of chronic gout.

In the absence of any contraindications, high-dose NSAIDs are the first-line treatment for acute gout. Naproxen 750mg as a stat dose followed by 250 mg TDS is a commonly used and effective regime.

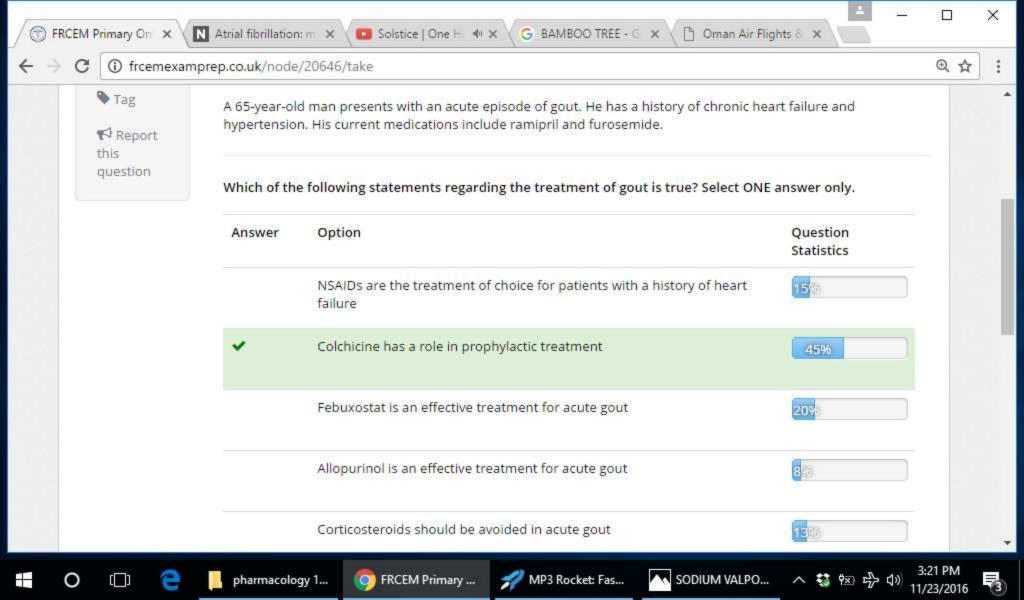
Aspirin should not be used in gout as it reduces the urinary clearance of urate and interferes with the action of urosuric agents. Naproxen, Diclofenac or Indomethacin are more appropriate choices.

Allopurinol is used prophylactically, preventing future attacks by reducing serum uric acid levels. It should not be started in the acute phase as it increases the severity and duration of symptoms.

Colchicine acts on the neutrophils, binding to tubulin to prevent neutrophil migration into the joint. It is as effective as NSAIDs in relieving acute attacks. It also has a role in prophylactic treatment if Allopurinol is not tolerated.

NSAIDs are contraindicated in heart failure as they can cause fluid retention and congestive cardiac failure. Colchicine is the preferred treatment in patients with heart failure or those who are intolerant of NSAIDs.

Finish



The diagnosis in this case is clearly that of gout. The European League Against Rheumatism (EULAR) guidelines for diagnosis state that the development of acute pain in a joint which becomes swollen, tender and erythematous and which reaches its crescendo over a 6-12 hour period is highly suggestive of crystal arthropathy.

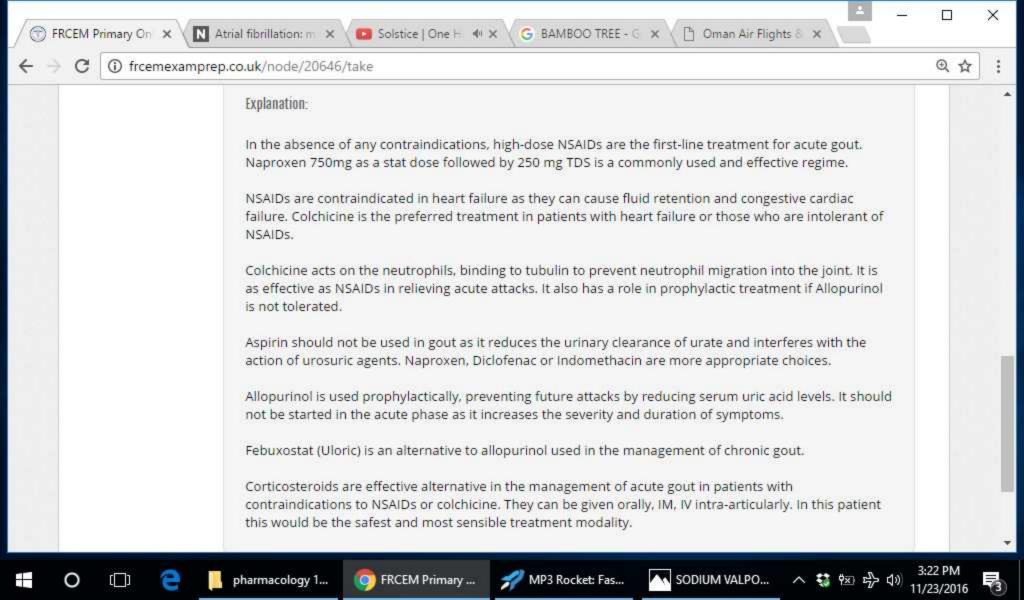
There is little benefit in checking serum urate levels to confirm hyperuricaemia prior to initiating treatment in acute attacks of gout and treatment should not be delayed. Although they can be helpful in monitoring response to treatment they often decrease during an acute attack and can be normal. If levels are checked and are normal during the attack they should be repeated once the attack has resolved.

The first-line treatment for acute attacks of gout is non-steroidal anti-inflammatory drugs (NSAIDs), such as naproxen. NSAIDs should, however, be used with caution in patients with a history of hypertension. Given that this patient has had difficulty controlling his blood pressure and remains hypertensive it would be prudent to avoid them in this case.

Colchicine is an effective alternative to gout, although it is somewhat slower to take effect. It is often used in patients with contraindications to NSAIDs, such as in patients with hypertension and those with a history of peptic ulcer disease. Colchine in addition to having anti-inflammatory effects can also have effects on the bone marrow and cause both neutrophilia and thrombocytopenia. It is therefore contraindicated in patients with blood disorders, such as in this case.

Allopurinol should not be used during an acute attack of gout as it can both prolong the attack and precipitate a further acute attack. In patients already established on allopurinol, it should be continued and the acute attack treated as normal with NSAIDs, colchicine or corticosteroids as appropriate.

Corticosteroids are effective alternative in the management of acute gout in patients with contraindications to NSAIDs or colchicine. They can be given orally, IM, IV intra-articularly. In this patient this would be the safest and most sensible treatment modality.



Supraventricular tachycardia (SVT) is the most common non-arrest arryhthmia during childhood and is the most common arrhythmia that produces cardiovascular instability during infancy.

The current APLS guidelines recommend that if the patient has no features of shock and remains haemodynamically stable then vagal maneovres should be attempted initially. If this is unsuccessful then:

- An initial dose of 100 mcg/kg of adenosine should be given.
- After two minutes another dose of 200 mcg/kg adenosine should be given is the child remains in stable SVT
- . After a further two minutes another dose of 300 mcg/kg adenosine should be given

If the child remains in stable SVT despite these measures then the guidelines recommend that following be considered:

- Adenosine 400-500 mcg/kg
- Synchronous DC shock
- Amiodarone

Amiodarone, if given, should be administered initially at a dose of 5-10 mg/kg over 20 minutes to 2 hours, then by continuous infusion 300 mcg/kg/hour increased according to response by 1.5 mg/kg/hour. The infusion rare should not exceed 1.2 g in 24 hours.

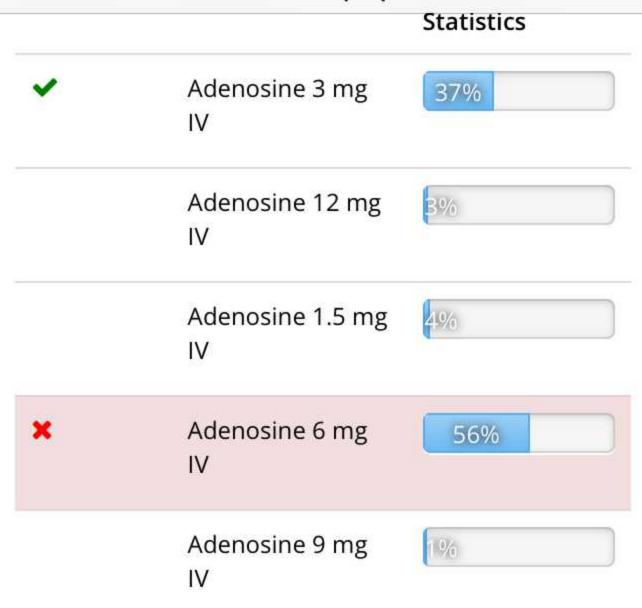
This child weighs 20 kg and has already received one dose of adenosine. He should therefore receive a second dose of 200 mcg/kg, which is 4 mg.



# 4:36 PM

91%

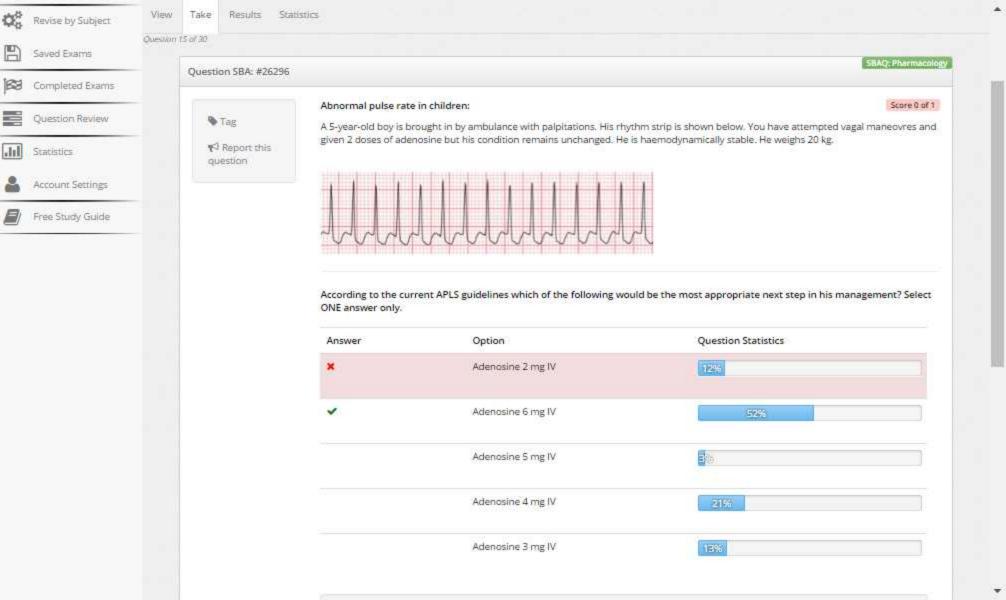
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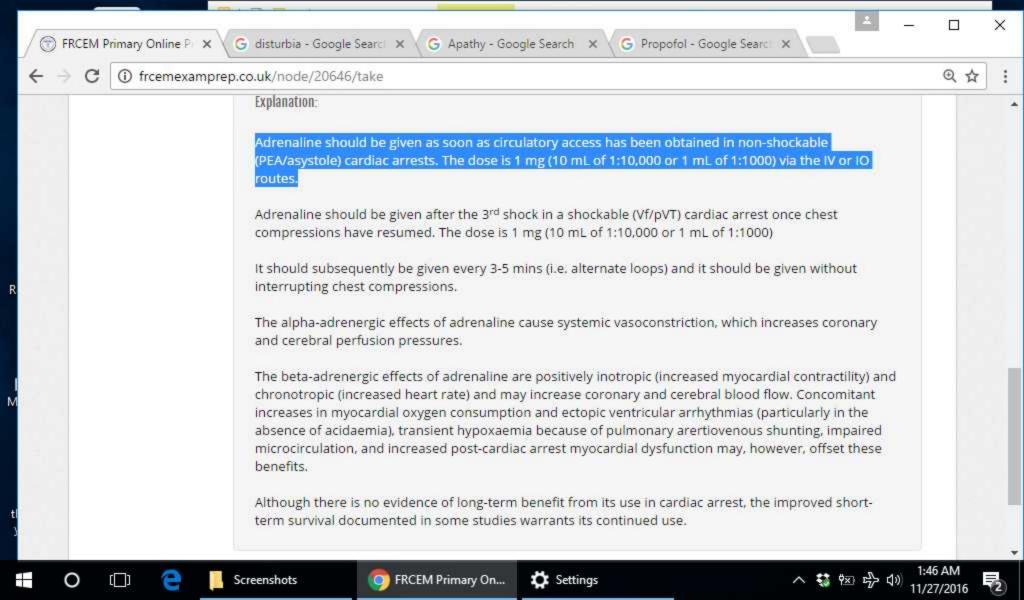
# **Explanation:**

Adenosine is administered by a rapid IV bolus, followed by a saline flush. The standard initial adult dose is 6 mg, followed if necessary by a 12 mg, and then a further 12 mg bolus at 1-2 minute intervals until an effect is observed.

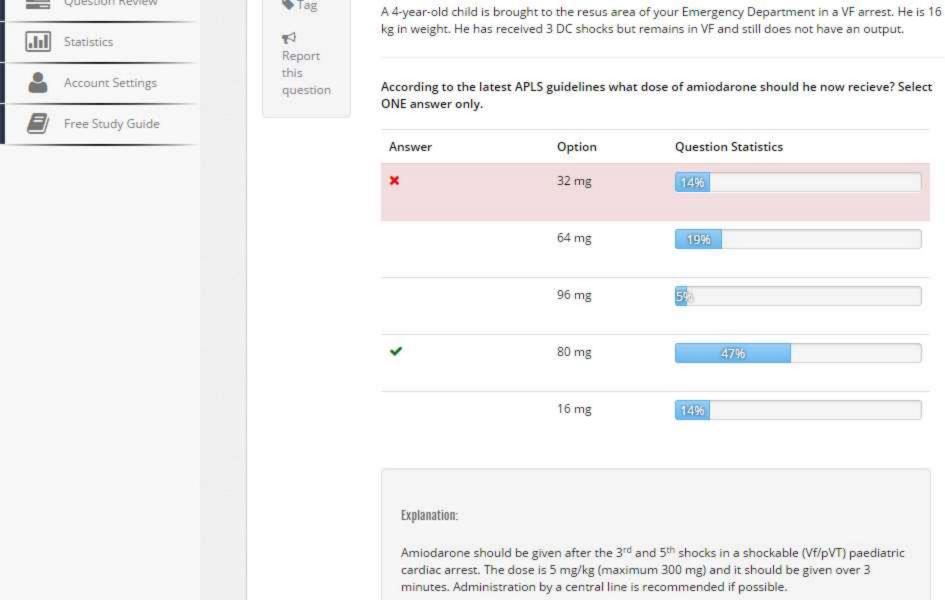
Patients with a heart transplant, however, are very sensitive to the effects of adenosine and should receive a reduced initial dose of 3mg, followed by 6 mg and then 12 mg.



Explanation: Adenosine is a purine nucleoside that is primarily used in the diagnosis and treatment of paroxysmal supraventricular tachycardia. It acts by stimulating A1-adenosine receptors and opening acetylcholine-sensitive potassium channels. This hyperpolarizes the cell membrane in the atrio-ventricular (AV) node and, by inhibiting the calcium channels, slows conduction in the AV node. Adenosine is administered by a rapid IV bolus, followed by a saline flush. The initial adult dose is 6 mg, followed if necessary by a 12 mg, and then a further 12 mg bolus at 1-2 minute intervals until an effect is observed. Adenosine has a very short half-life of less than 10 seconds and acts rapidly within 10 seconds. The duration of actions is 10-20 seconds. Because of the short half-life any side effects experienced are generally very short lived. These include: · Sense of 'impending doom' · Facial flushing Dyspnoea Chest discomfort Metallic taste Contra-indications to the use of adenosine include: 2<sup>nd</sup> or 3<sup>rd</sup> degree AV block · Sick sinus syndrome · Long QT syndrome · Severe hypotension · Decompensated heart failure Chronic obstructive lung disease Asthma Patients with a heart transplant are very sensitive to the effects of adenosine and should receive a reduced initial dose of 3mg, followed by 6 mg and then 12 mg. The effects of adenosine are potentiated by dipyrimadole and the dose should be reduced in patients taking



Explanation:  Corneal microdeposits are almost universally present (over 90%) in individuals taking amiodarone for longer than 6 months, especially at does greater that 400 mg/day. The deposits typically do not cause any symptoms but about 10% of patients complain of seeing a 'bluish halo'.  Amiodarone also has other effects on the eye, but these are much rarer occurring in or 1-2% of patients:  Optic neuropathy  Non-arteritic anterior ischaemic optic neuropathy (N-AION)  Optic disc swelling  Visual field defects		Digoxin	9%
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Optic neuropathy     Non-arteritic anterior ischaemic optic neuropathy (N-AION)     Optic disc swelling	Corneal microo amiodarone fo deposits typica	r longer than 6 months, especi lly do not cause any symptoms	ally at does greater that 400 mg/day. The
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150 mg	9%	
250 mg	1970	

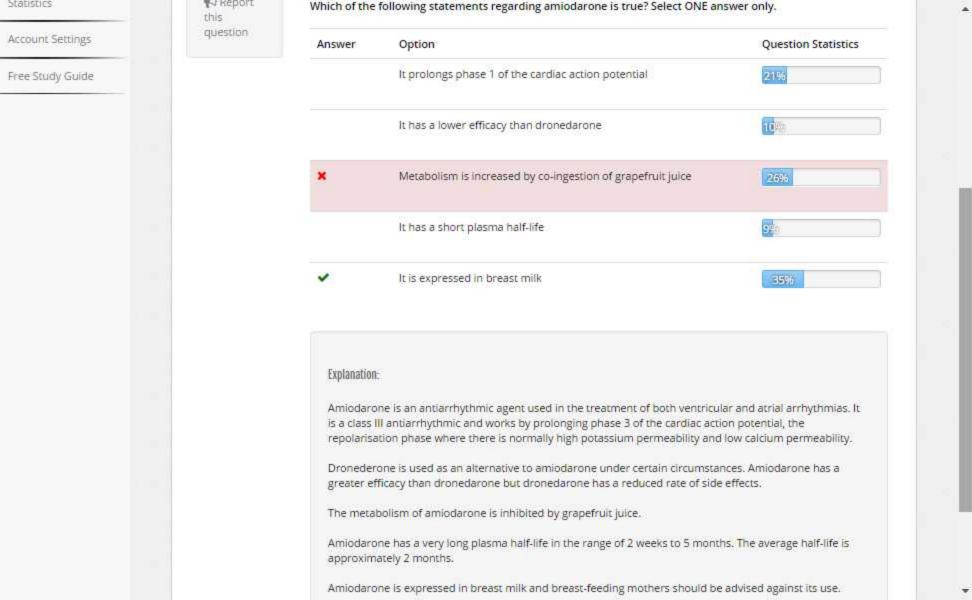
Amiodarone should be given after the  $3^{rd}$  shock in a shockable (Vf/pVT) cardiac arrest during chest compressions. The dose is 300 mg as an IV bolus diluted in 5% dextrose to a volume of 20 mL.

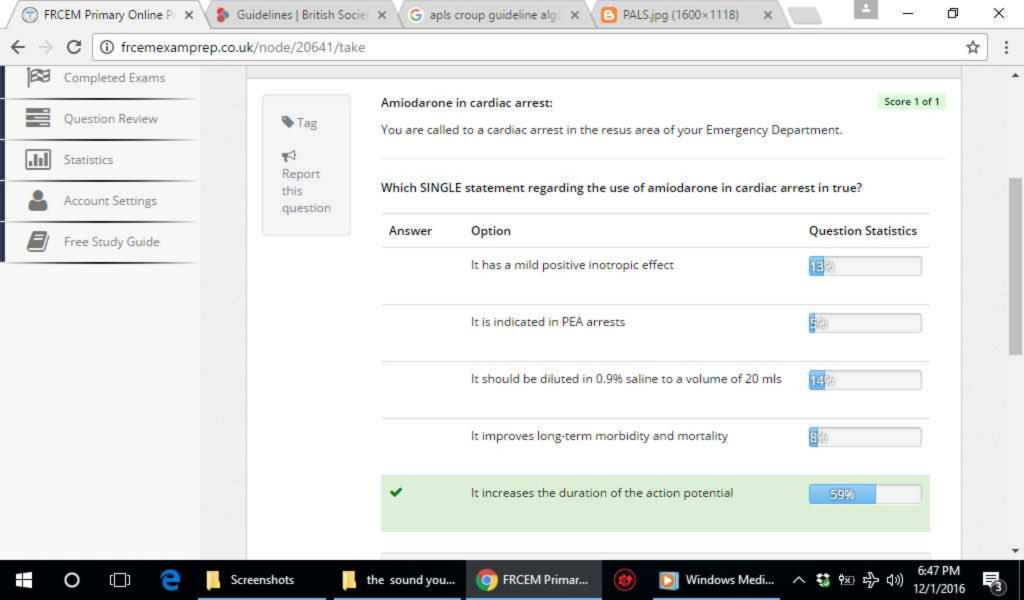
A further dose of 150 mg should be given if VF/pVT persists after 5 defibrillation attempts.

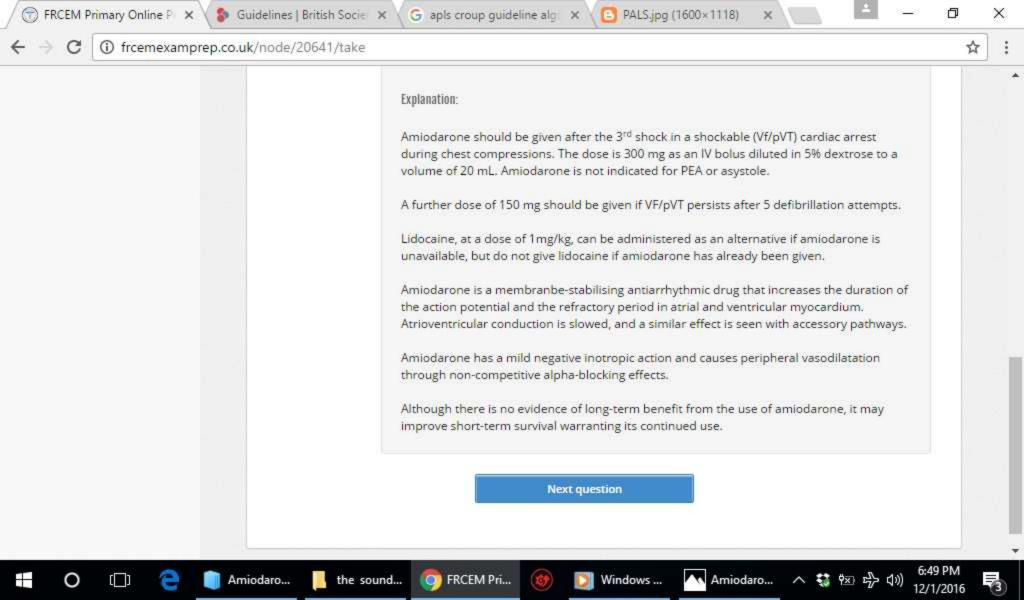
Amiodarone is not indicated for PEA or asystole.

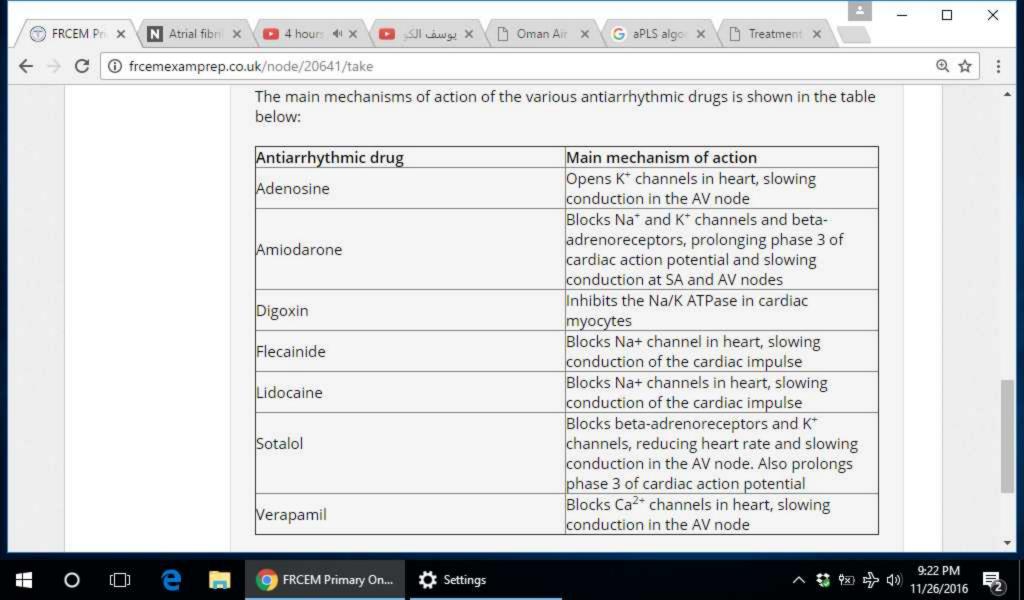
Lidocaine, at a dose of 1mg/kg, can be administered as an alternative if amiodarone is unavailable, but do not give lidocaine if amiodarone has already been given.

Next question









Score 0 of 1



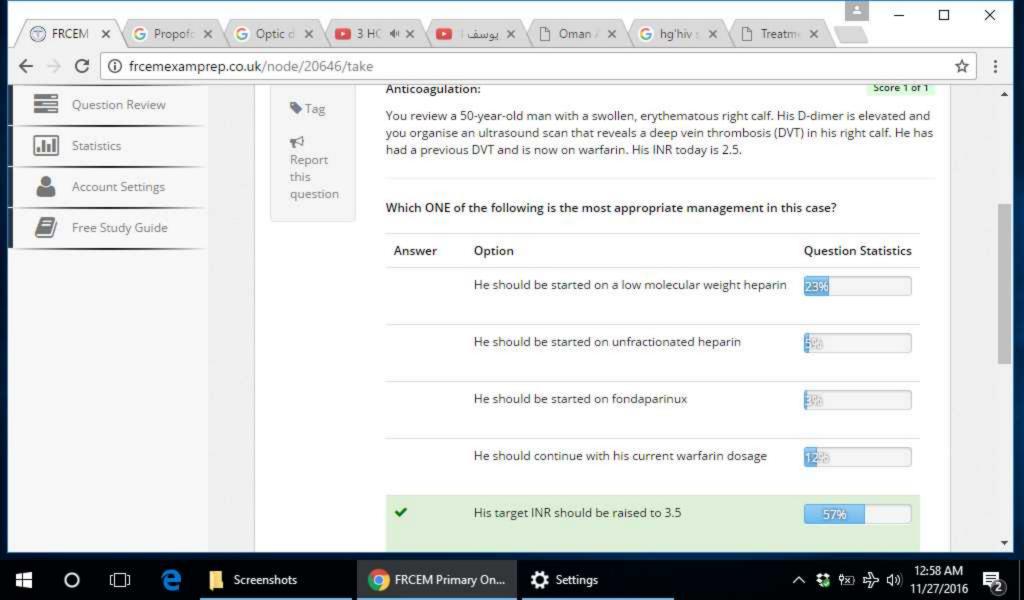
Report this question

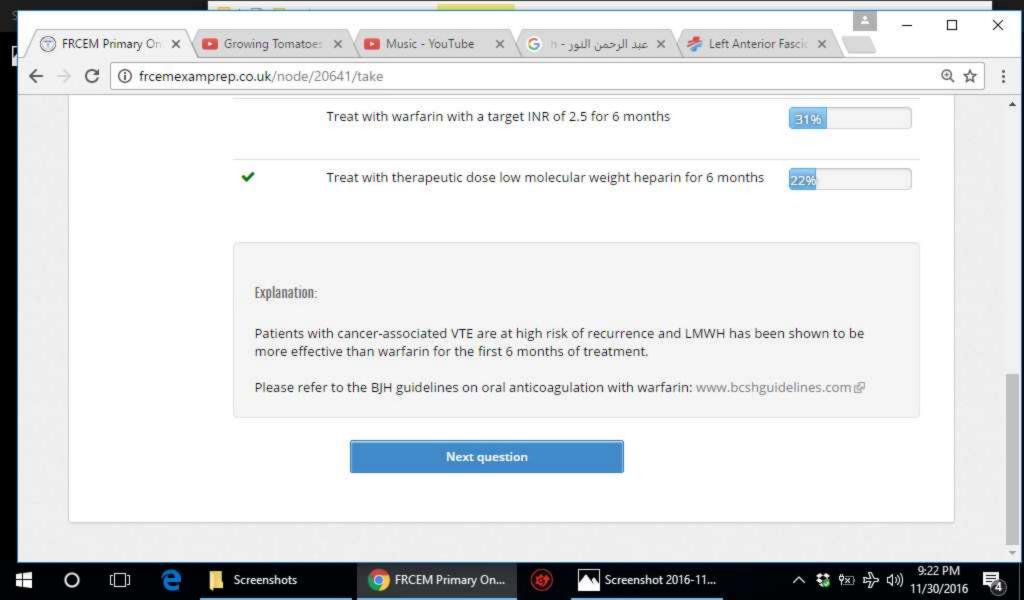
## Anticoagulation:

You review a 70-year-old man with a diagnosis of prostate cancer. He presents with left leg swelling, erythema and tenderness. You organise an ultrasound scan of his leg, which reveals a large proximal deep vein thrombosis (DVT). He has had no previous episodes of venous thromboembolism and has no other past medical history of note.

What is the most appropriate management strategy in this case? Select ONE answer only.

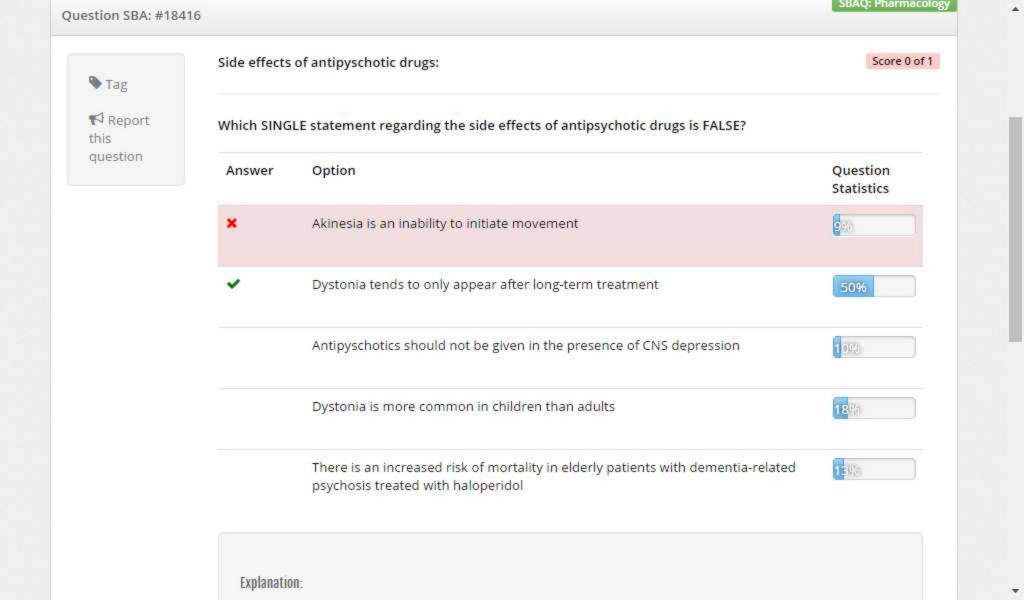
Answer	Option	Question Statistics
	Treat with therapeutic dose low molecular weight heparin for 3 months	13%
×	Treat with warfarin with a target INR of 2.5 for 3 months	26%
	Treat with warfarin with a target INR of 3.5 for 6 months	<b>8</b> 96
	Treat with warfarin with a target INR of 2.5 for 6 months	31%
<b>~</b>	Treat with therapeutic dose low molecular weight heparin for 6 months	22%





There are numerous specific antidotes available for specific poisons and overdoses. Some of these are outlined in the table below:

Poison	Antidote	
Benzodiazepines	Flumazenil	
Beta-blockers	Atropine Glucagon Insulin	
Carbon monoxide	Oxygen	
Cyanide	Hydroxocobalamin Sodium nitrite Sodium thiosulphate	
Digoxin	Digoxin-specific antibody Fab fragments (Digibind)	
Ethylene glycol	Ethanol Fomepizole	
Heparin	Protamine sulphate	
Iron salts	Desferrioxamine	
Isoniazid	Pyridoxine	
Methanol	Ethanol Fomepizole	
Opioids	Naloxone	
Organophosphates	Atropine Pralidoxime	
Paracetamol	Acetylcysteine Methionine	
Sulphonylureas	Glucose Octreotide	
Thallium	Prussian blue	
Warfarin	Vitamin K Fresh frozen plasma (FFP)	



Extrapyramidal side effects occur most commonly with the piperazine phenothiazines (fluphenazine, prochlorperazine and trifluoperazine) and butyrophenones (benperidol and haloperidol). Haloperidol is the most common causative antipsychotic drug.

Tardive dyskinesia (rhythmic, involuntary movements of tongue, face and jaw) usually develops after longterm treatment or with high dosage. It is the most serious manifestation of extrapyramidal symptoms as it may be irreversible on withdrawing the causative drug and treatment is generally ineffective.

Dystonia (abnormal face and body movements) is more common in children and young adults and tends to appear after only a few doses. Acute dystonia can be treated with procyclidine 5mg IV or benzatropine 2mg IV as a bolus.

Akathisia is characterized by an unpleasant sensation of restlessness. Akinesia is an inability to initiate movement.

There is increased cerebral sensitivity in renal impairment and reduced doses should be used.

There is an increased risk of mortality in elderly patients with dementia-related psychosis treated with haloperidol. This appears to be due to increased risk of cardiovascular events and infections such as pneumonia.

The contraindications to the use of antipsychotic drugs include:

- · Reduced conscious level / coma
- CNS depression
- Phaeochromocytoma

The current APLS algorithm for the treatment of the convulsing child is as follows:

#### Step 1 (5 minutes after start of convulsion):

In a child that has been convulsing for 5 minutes or more an initial dose of benzodiazepine should be given:

- Lorazepam 0.1 mg/kg should be given IV or IO if vascular access is available
- Buccal midazolam 0.5 mg/kg or rectal diazepam 0.5 mg/kg can be given as alternatives if no vascular access is available

#### Step 2 (10 minutes after start of step 1):

If the convulsion continues for a further 10 minutes a second dose of benzodiazepine should be given and senior help should be summoned.

#### Step 3 (10 minutes after start of step 2):

At this stage senior help is needed to reassess the child and advise on management. The following management is recommended:

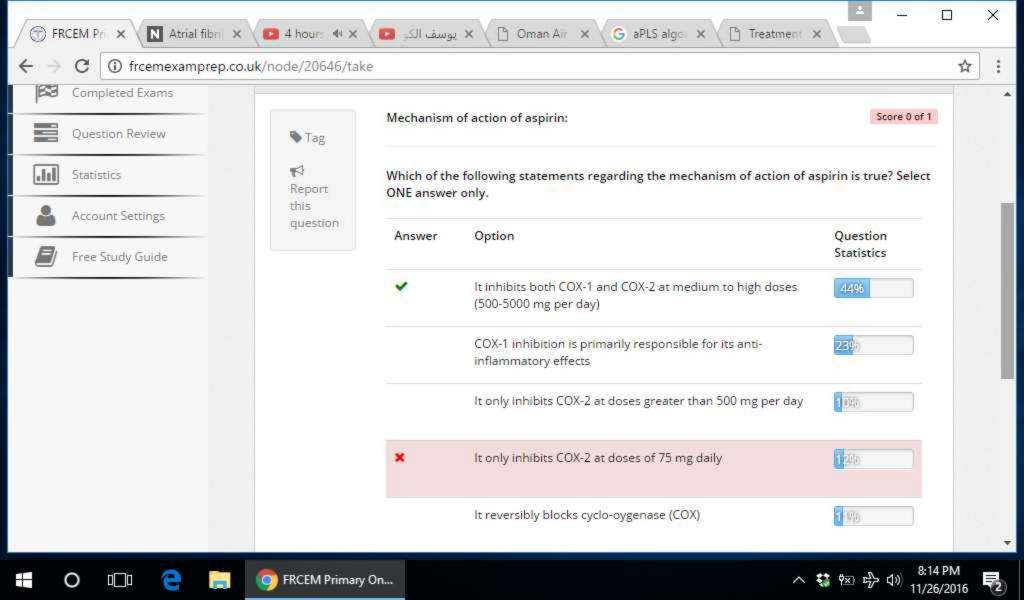
- If not already on phenytoin then a phenytoin infusion should be set up (20 mg/kg IV infusion over 20 minutes)
- If already taking phenytoin then phenobarbitone can be used in its place (20 mg/kg IV infusion over 20 minutes)
- Rectal paraldehyde can be considered at a dose of 0.8 ml/kg of the 50:50 mixture whilst preparing the infusion

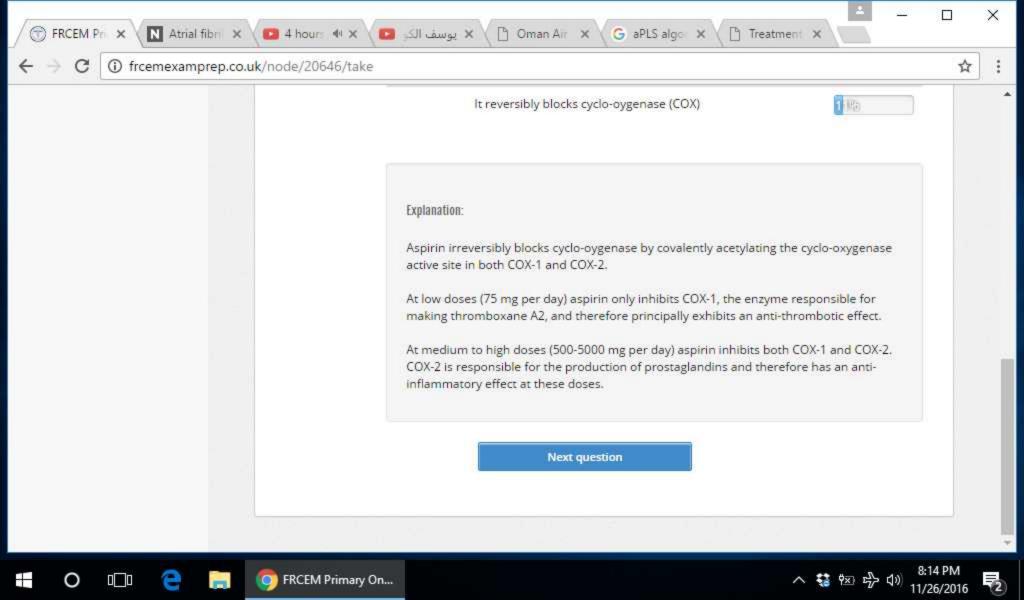
#### Step 4 (20 minutes after start of step 3):

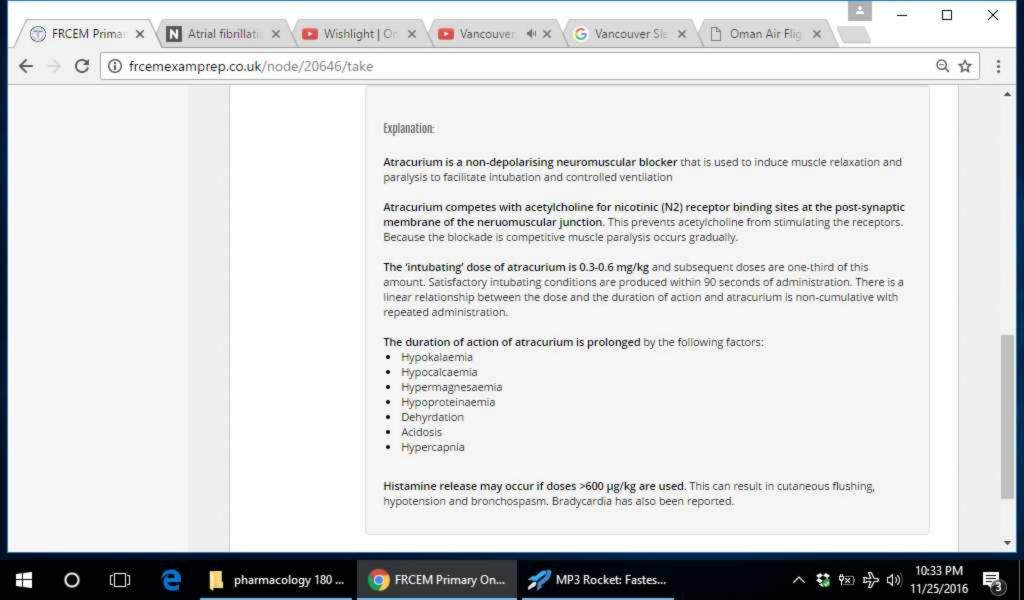
If the child is still convulsing at this stage then an anaesthetist must be present and a rapid sequence induction with thiopental is recommended.

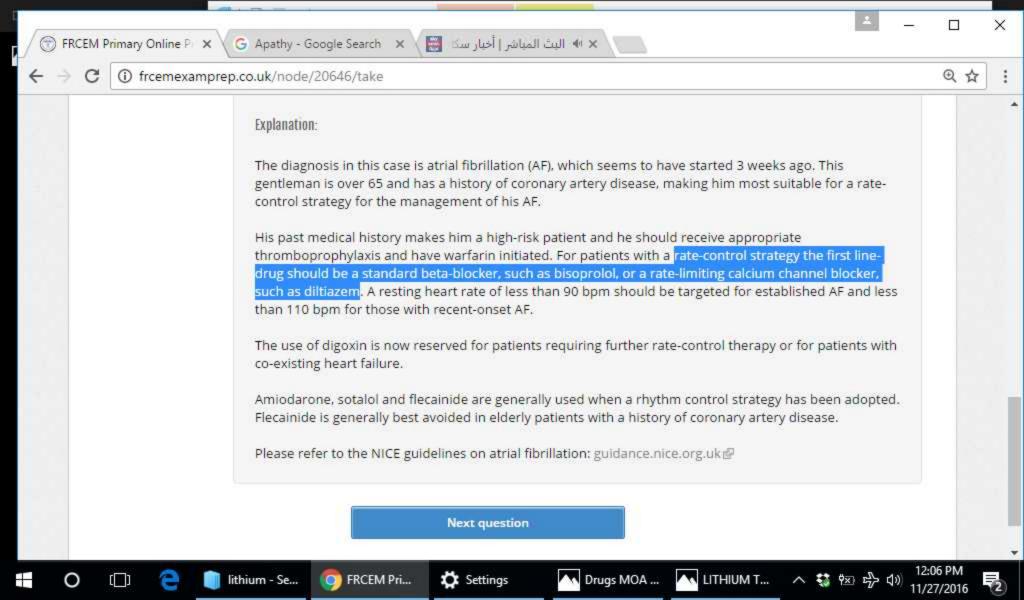
The following table summarises the doses of drugs commonly used in paediatric cardiac arrest:

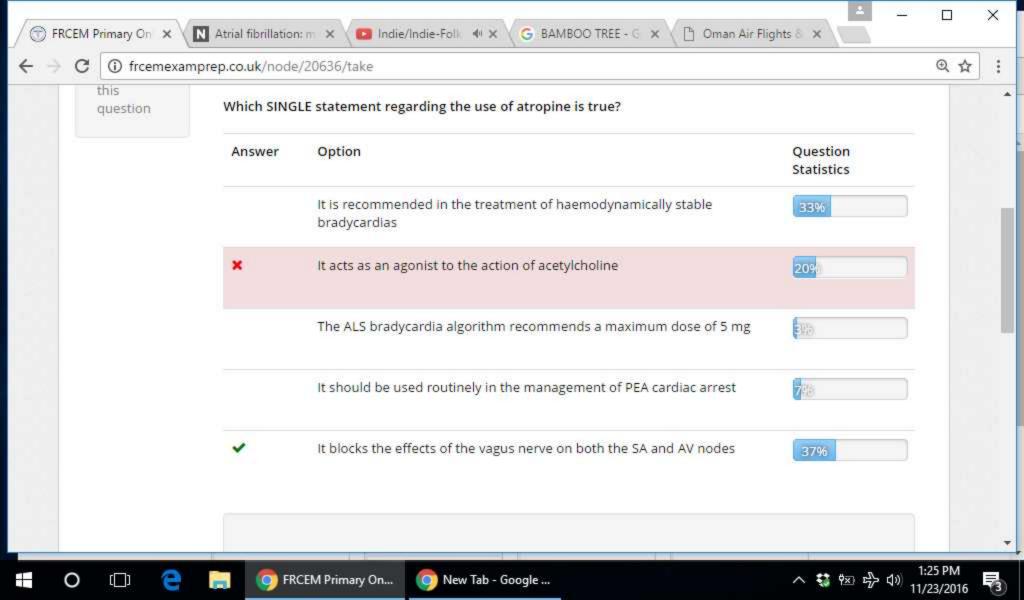
Drug	Dose	
Adrenaline (epinephrine) IV/IO	10 mcg/kg	
Adrenaline (epinephrine) ET bolus	100 mcg/kg	
Amiodarone IV infusion	5 mg/kg over 3 minutes (max 300 mg)	
Calcium gluconate 10%	0.3-0.5 ml/kg	
Lidocaine IV/IO	1 mg/kg (max 100 mg)	
Magnesium sulphate IV	25-50 mg/kg	
Sodium bicarbonate IV	1 ml/kg 8.4%	





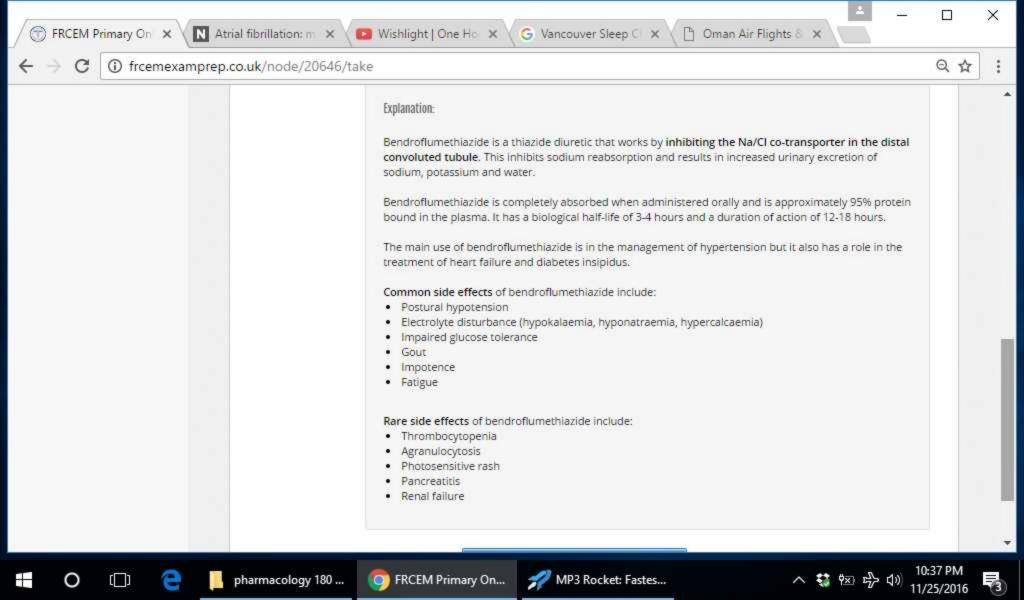


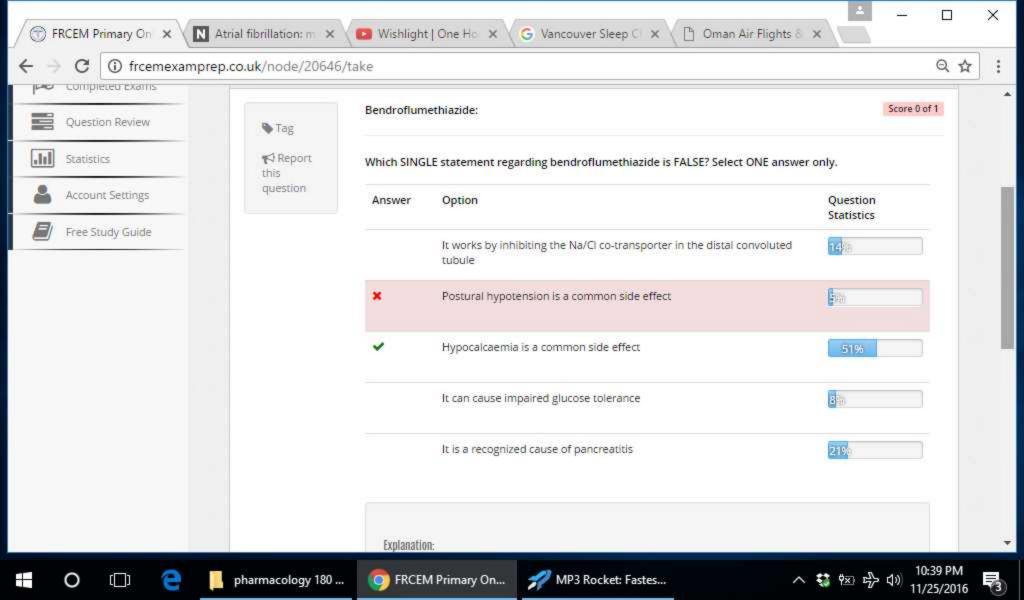




Atropine antagonises the action of the parasympathetic neurotransmitter acetylcholine at muscarinic receptors. It therefore blocks the effects of the vagus nerve on both the SA node and the AV node, increasing sinus automaticity and facilitating AV node conduction. The side effects of atropine are dose related and include: Dry mouth Nausea and vomiting Blurred vision Urinary retention Tachyarrhythmias It can also cause acute confusion and hallucinations, particularly in elderly patients. Atropine is indicated for sinus, atrial, or nodal bradycardia or AV block, when the haemodynamic condition of the patient is unstable because of the bradycardia. The ALS bradycardia algorithm recommends a dose of 500 mcg IV if any of the following adverse features are present: Shock Syncope Myocardial ischaemia Heart failure If this is unsuccessful further 500 mcg doses can be given at 3-5 minute intervals until a maximum dose of 3 mg is reached. Doses greater than 3 mg can cause paradoxical slowing of the heart rate. Asystole during cardiac arrest is usually caused by primary myocardial pathology rather than excessive vagal tone and there is no evidence that routine use of atropine is beneficial in the treatment of asystole or PEA. For this reason it no longer forms part of the non-shockable part of the ALS algorithm.

Explanation:







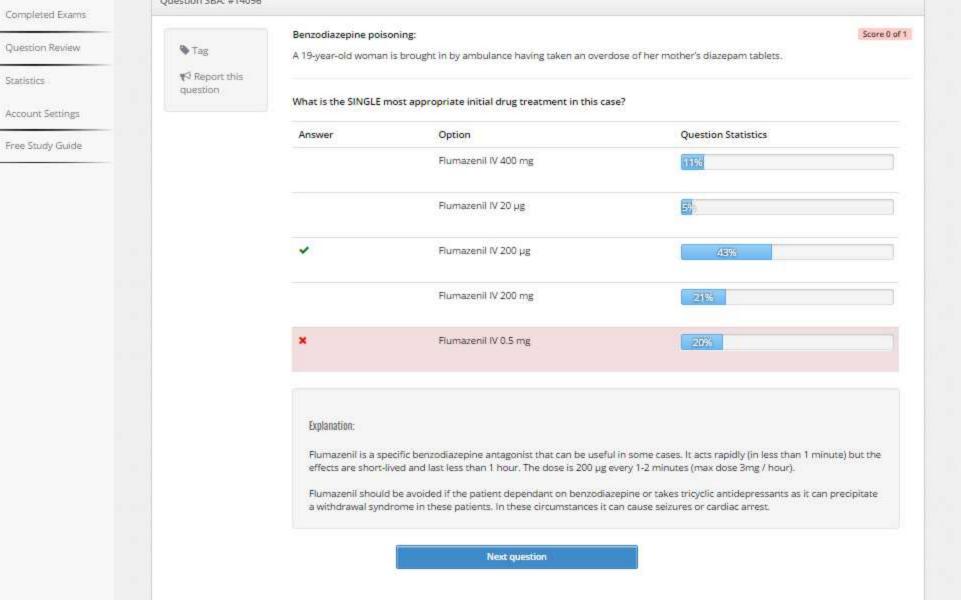
question

Which of the following is the mechanism of action of bendroflumethiazide? Select ONE answer only.

Answer	Option	Question Statistics
~	Inhibition of sodium reabsorption in the distal convoluted tubule	59%
	Inhibition of the Na/Cl co-transporter in the proximal convoluted tubule	17%
	Inhibition of the Na/K/2Cl symporter in the ascending loop of Henle	116
×	Creating an osmotic diuresis	Esh
	Inhibition of the Na/K/2Cl symporter in the descending loop of Henle	10.94

Explanation:

Bendroflumethiazide is a thiazide diuretic that works by inhibiting the Na/Cl co-transporter in the distal convoluted tubule. This inhibits sodium reabsorption and results in increased urinary excretion of sodium, potassium and water.



dil

Statistics

COLLECTIONS ECG LIBRARY TOX LIBRARY CCC TABLES TOP 100 EXAMS NOVEMBER 28, 2016

# Background

- Bifascicular block is the combination of RBBB with either LAFB or LPFB.
- . Conduction to the ventricles is via the single remaining fascicle.
- The ECG will show typical features of RBBB plus either left or right axis deviation.
- RBBB + LAFB is the most common of the two patterns.
- Bifascicular block is a sign of extensive conducting system disease, although the risk of progressing to complete heart block is thought to be relatively low (1% per year in one cohort study of 554 patients).

NB. Some authors also consider LBBB to be a 'bifascicular block', because both fascicles of the left bundle branch are blocked

# TechTool Thursday 070 D-Eye Research and Reviews in the Fastlane 161 Master the bronchoscope! LITFL Review 258 Funtabulously Frivolous Friday Five 166 Research and Reviews in the Fastlane 160

### Main Causes

- Ischaemic heart disease (40-60% cases)
- Hypertension (20-25%)
- Aortic stenosis
- Anterior MI (occurs in 5-7% of acute AMI)
- Primary degenerative disease of the conducting system (Lenegre's / Lev's disease)
- Congenital heart disease
- Hyperkalaemia (resolves with treatment)







with COPD

# **Explanation:**

Bisoprolol is a cardioselective beta-blocker that selectively blocks b<sub>1</sub> adrenergic receptors. It is commonly used in the management of hypertension, atrial fibrillation (for which it is now first-line therapy), and heart failure.

It has good oral bioavailability and peak plasma concentrations occur within 2 to 4 hours of administration. Its plasma half-life is 9-12 hours.

Bisoprolol is an effective and safe antihypertensive medication and has a stronger antihypertensive effect than propranolol and metoprolol.

Bisoprolol can be used in patients with COPD due to its b<sub>1</sub> selectivity, although it should be used with caution.

The CIBIS-II study showed that bisoprolol reduced mortality in all NYHA grades of heart failure.

### Explanation:

This patient has a bradycardia caused by a 2:1 fixed ratio block. Fixed ratio blocks occur when there is a 2nd degree heart block with a fixed ratio of P waves to QRS complexes. In this case there is a 2:1 block as there are two P waves for every one QRS complex.

Fixed ratio blocks can be due to either Mobitz I or Mobitz II atrioventricular block. It is not always easy to determine which of these is the underlying cause of the fixed ratio block but the QRS complex provides important clues.

### Generally speaking:

- Mobitz I conduction usually produces narrow QRS complexes as the block is located at the level of the AV node. Mobitz I blocks tend to improve with atropine and have an overall more benign prognosis.
- Mobitz II conduction usually produces broad QRS complexes (often in the context of a pre-existing LBBB). These tend to be unresponsive to atropine and are more likely to progress to complete heart block or asystole.

Atropine is indicated for sinus, atrial, or nodal bradycardia or AV block, when the haemodynamic condition of the patient is unstable because of the bradycardia.

The ALS bradycardia algorithm recommends a dose of atropine 500 mcg IV if any of the following adverse features are present:

- Shock
- Syncope
- Myocardial ischaemia
- · Heart failure

If this is unsuccessful further 500 mcg doses can be given at 3-5 minute intervals until a maximum dose of 3 mg is reached. Doses greater than 3 mg can cause paradoxical slowing of the heart rate.

Other interim measures suggested by ALS bradycardia algorithm include:

- Transcutaneous pacing
- · Isoprenaline infusion 5 mcg/min
- · Adrenaline infusion 2-10 mcg/minutes
- Alternative drugs (aminophylline, dopamine, glucagon, glycopyrrolate)

# **Explanation:**

The ALS bradycardia algorithm recommends a dose of 500 mcg IV if any of the following adverse features are present:

- Shock
- Syncope
- Myocardial ischaemia
- Heart failure

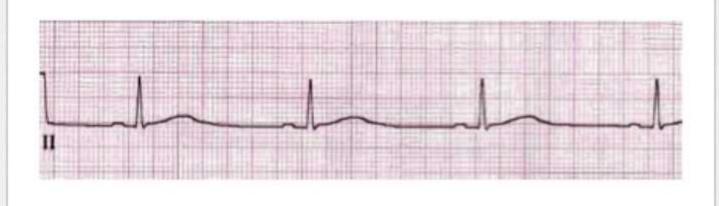
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Other interim measures suggested by ALS bradycardia algorithm include:

- Transcutaneous pacing
- Isoprenaline infusion 5 mcg/min
- Adrenaline infusion 2-10 mcg/minutes
- Alternative drugs (aminophylline, dopamine, glucagon, glycopyrrolate)

Glucagon is recommended if the bradycardia is caused by beta-blocker or calcium-channel blockers, and would therefore be the most appropriate choice in this case. The recommended dose is 2-10 mg IV in adults and 50-150 mcg/kg in children, followed by an intravenous infusion of 50 mcg/kg/hour.

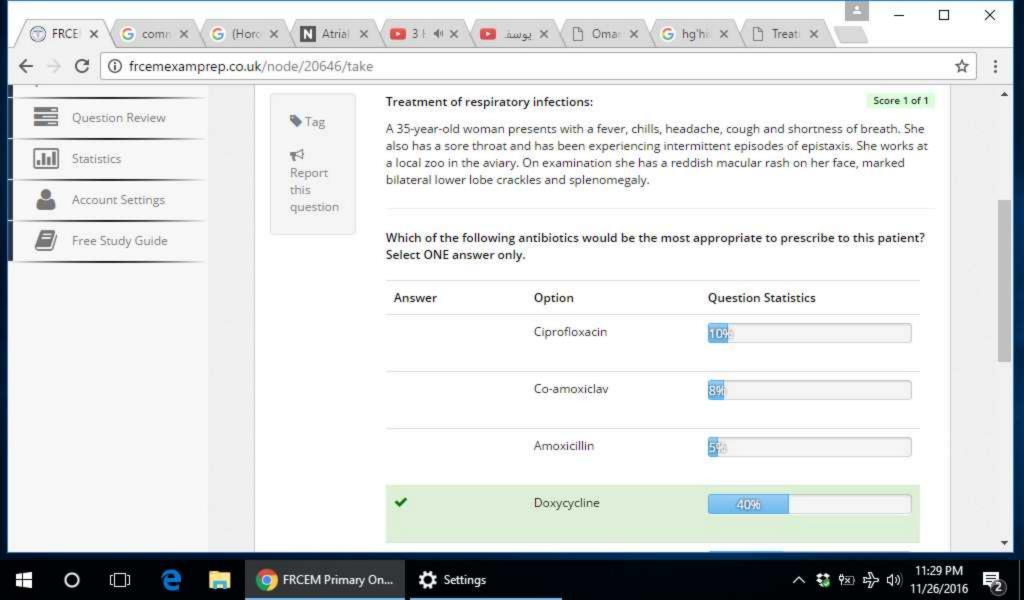
A 40-year-old woman has taken an overdose of atenolol. She is clammy and hypotensive, with a blood pressure of 70/45 mmHg. She has been given three 500 mcg doses of atropine but remains bradycardic and dizzy. Her heart rate is between 35 and 40 bpm and her rhythm strip is shown below:



According to the ALS bradycardia algorithm which of the following would be the most appropriate next step in her management? Select ONE answer only.

Answer	Option	Question Statistics
	Give atropine 3 mg as an IV bolus	9%
	Give 1 mg IV bolus of adrenaline	13%
×	DC synchonrised shock	13%
~	Give glucagon 2 mg	62%

Observe on a cardiac



# frcemexamprep.co.uk

# **Explanation:**

Cefuroxime and the other cephalosporin antibiotics are ß-lactam antibiotics and are bactericidal. Like the penicillins they produce their antimicrobial action by preventing crosslinkage between the linear peptidoglycan polymer chains that make up the bacterial cell wall. They therefore inhibit cell wall synthesis.

An overview of the different mechanisms of action of the various types of antimicrobial agents is shown below:

Mechanism of action	Examples
Inhibition of cell wall synthesis	Penicillins Cephalosporins Vancomycin
Disruption of cell membrane function	Polymyxins Nystatin Amphotericin B
Inhibition of protein synthesis	Macrolides Aminoglycosides Tetracyclines Chloramphenicol
Inhibition of nucleic acid synthesis	Quinolones Trimethoprim 5-nitroimidazoles Rifampicin
Anti-metabolic activity	Sulfonamides Isoniazid

the likelihood of group A beta haemolytic Streptococcus (GABHS) infection in adult patients complaining of a sore throat. A study published in the BMJ in 2013 looked at whether they could be applied to children.

The Centor Criteria are a set of criteria that were originally developed as a tool to identify

As a consequence of this study the modified criteria were developed, which add in the patient's age and can be used to assess children over the age of 2. Scores may range from -1 to +5

Patients are judged on the following criteria, with one point for each positive criterion:

- History of a fever (Temp > 38°C) · Exudate or swelling on tonsils
- · Tender or swollen anterior cervical lymph nodes
- · Absence of cough

The patient's age is scored as follows:

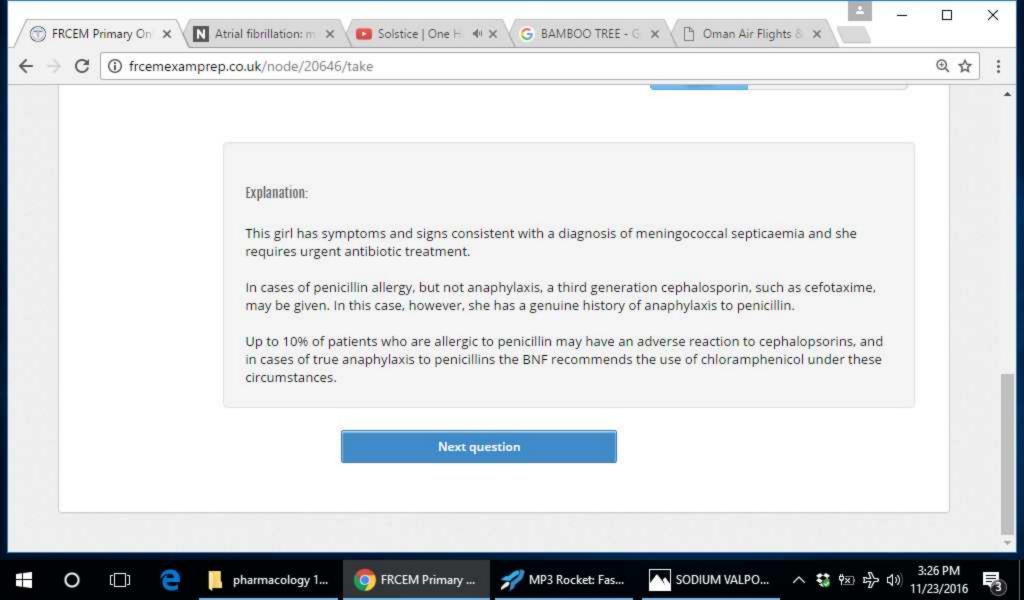
- 3-14 years = +1 point
- 15-44 years = 0 points
- > 45 years = -1 point

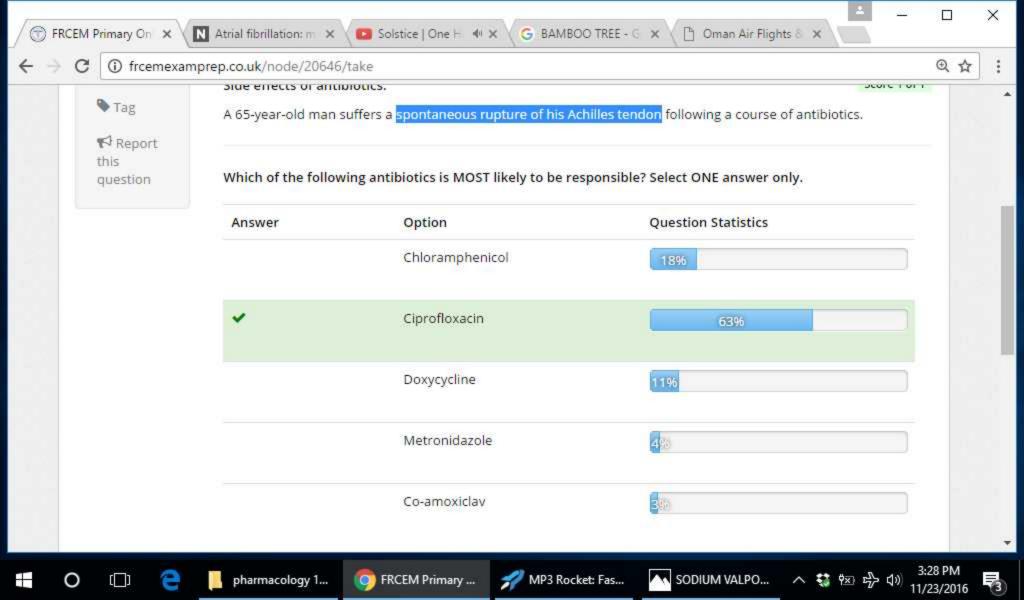
The score can then be used to guide management as follows:

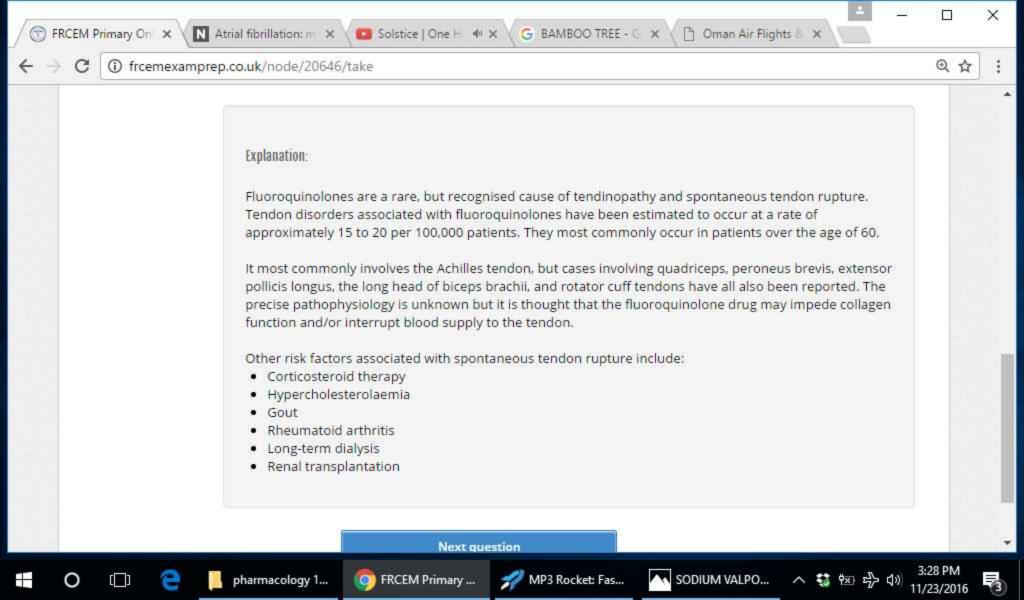
- -1 to +1 points no antibiotics or throat culture is necessary
- 2 to 3 points patients should receive a throat culture and treat with an antibiotic if the culture is positive
- 4 to 5 points patients should be treated empirically with antibiotics

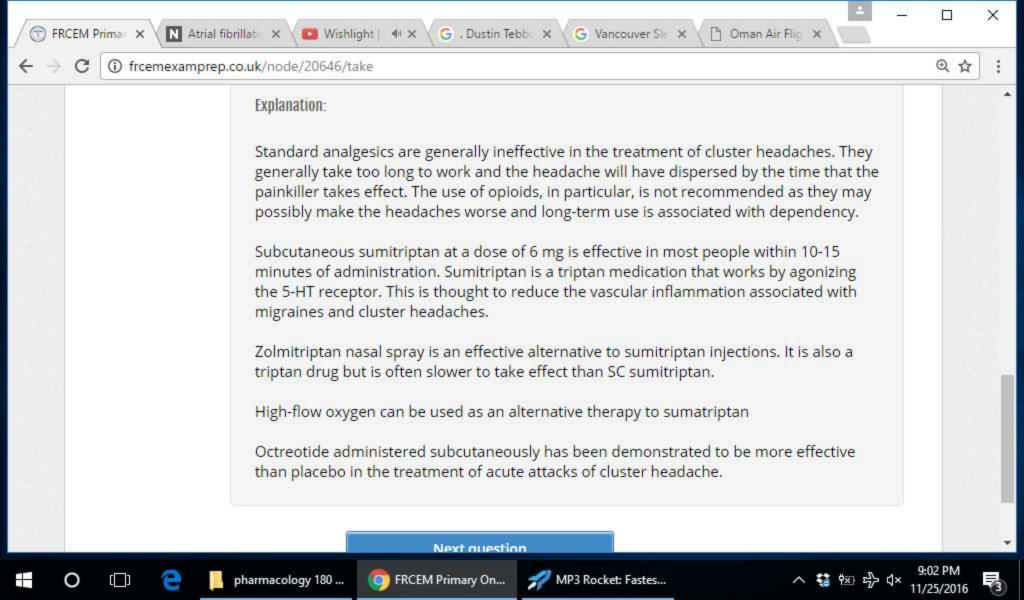
This girl has a score of 4 points and should therefore be treated empirically with antibiotics. The current SIGN guidelines recommend a course of oral penicillin V (phenoxymethylpenicillin) for 10 days as the first choice antibiotic. For a child aged 9 the appropriate dose would be 250 mg of penicillin. Erythromycin is suitable alternative for patients with a penicillin allergy.

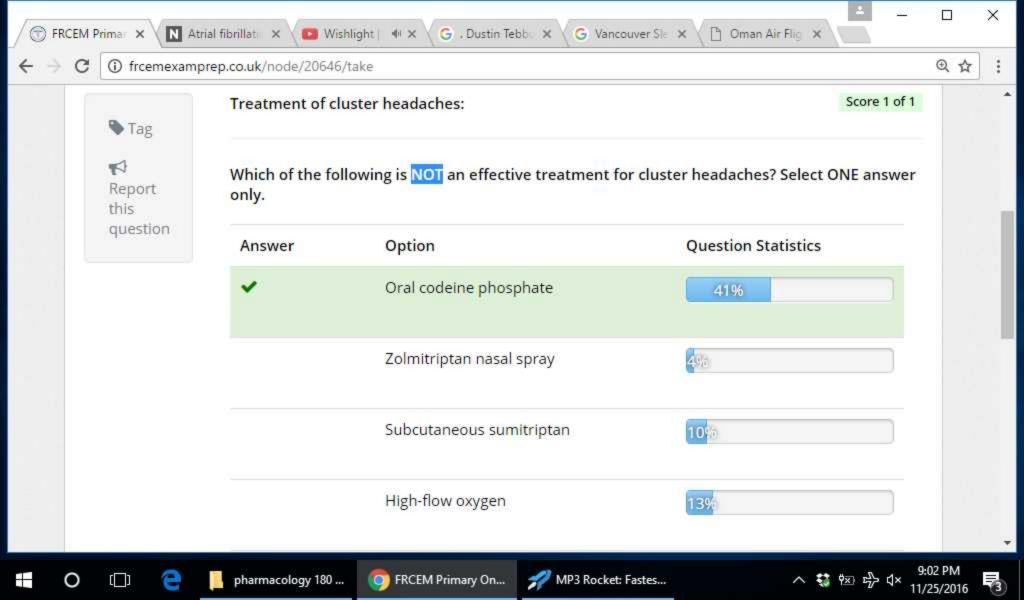
	Ibuprofen	34%	
	Nitrofurantoin	12%	
Explanation:			
Isoniazid can cause an ac	ute, dose-dependant, hepat	itis but it is not a recognised cause of	cholestatic
aundice.			
Deure which extre choles	tatic incodice lockeds:		
<ul> <li>Drugs which cause choles</li> <li>Nitrofurantoin</li> </ul>	static jaundice include:		
Erythromycin			
Cephalosporins			
<ul> <li>Verapamil</li> </ul>			
NSAIDs			
<ul> <li>ACE inhibitors</li> </ul>			
<ul> <li>Tricyclic antidepressa</li> </ul>	nts		
Phenytoin			
<ul> <li>Azathioprine</li> </ul>			
<ul> <li>Carbemazepine</li> </ul>			
<ul> <li>Oral contraceptive pill</li> </ul>	5		
<ul> <li>Diazepam</li> </ul>			
Ketoconazole			
Tamoxifen			
7			
	Next question		
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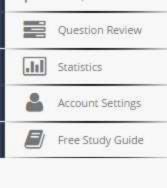














Tag.

this question

\* Report

Score 0 of 1 Drug treatment of croup:

A 2-year-old child is brought in to the paediatric area of your Emergency Department. She has a harsh, barking cough and stridor. The sister in charge of the area has requested that you prescribe oral dexamethasone as part of their croup management protocol.

According to the APLS guidelines what is the suggested maximum single dose of dexamethasone for croup? Select ONE answer only.

Answer	Option	Question Statistics
×	6 mg	25%
	8 mg	15%
	15 mg	5%
~	12 mg	41%
	10 mg	14%

Explanation:

No definite standard dose for the use of dexamethasone has currently been agreed in the UK. The APLS guidelines, however, recommend a dose of 150 mcg/kg, with a suggested maximum single dose of 12 mg.

3390

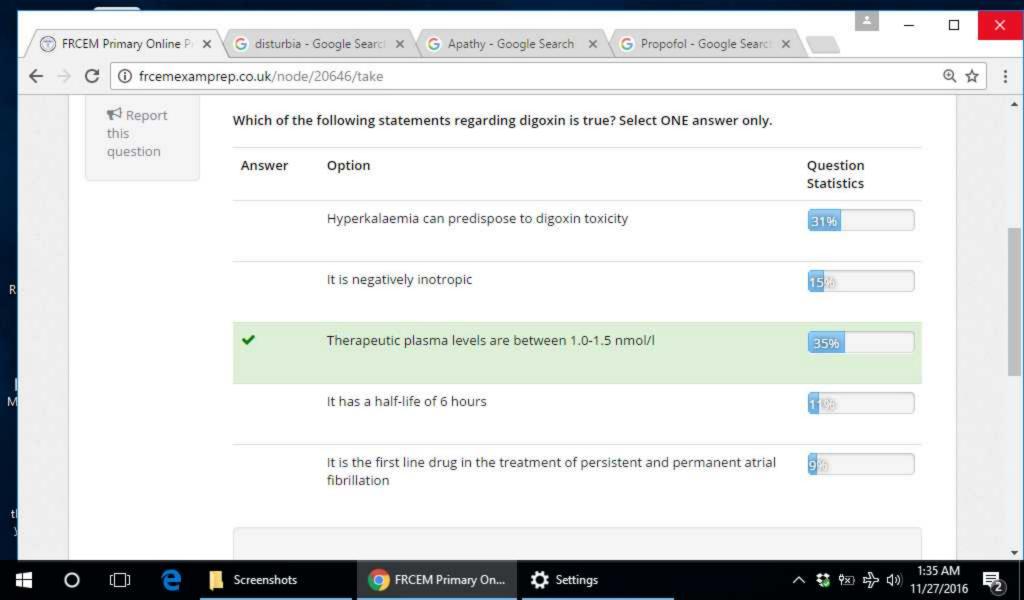
## **Explanation:**

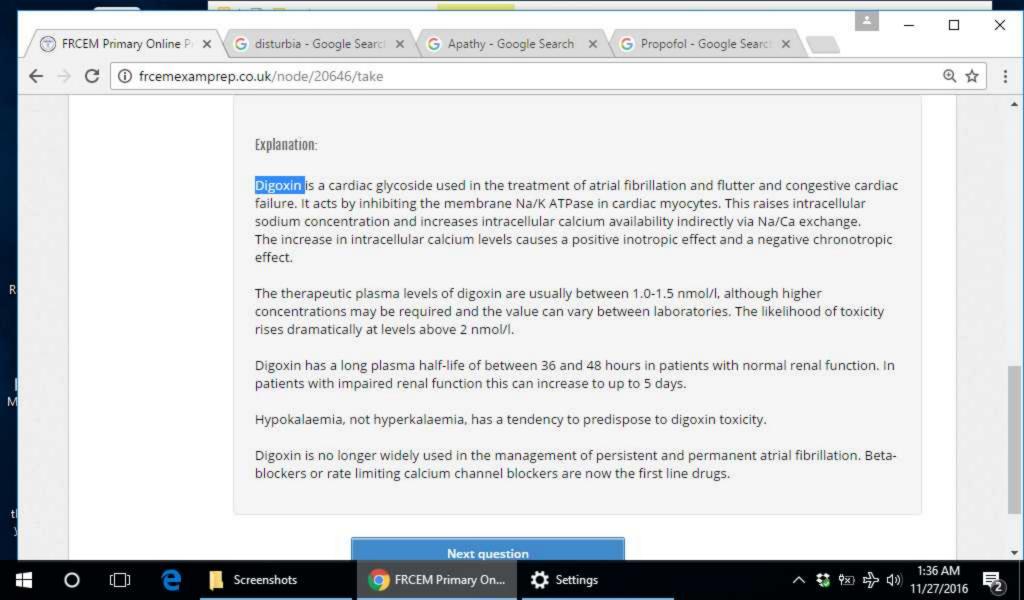
Hyperpyrexia and hypoglycaemia are seen more commonly in children than adults as a result of salicylate poisoning.

Other clinical features, that are seen in both adults and children, include:

- Nausea and vomiting
- Tinnitus
- Deafness
- Sweating and dehydration
- Hyperventilation
- Cutaneous flushing

Xanthopsia is associated with digoxin toxicity, not salicylate poisoning.





### Explanation:

Digoxin is a cardiac glycoside used in the treatment of atrial fibrillation and flutter and congestive cardiac failure. It acts by inhibiting the membrane Na/K ATPase in cardiac myocytes. This raises intracellular sodium concentration and increases intracellular calcium availability indirectly via Na/Ca exchange.

The increase in intracellular calcium levels causes a positive inotropic effect and a negative chronotropic effect.

The therapeutic plasma levels of digoxin are usually between 1.0-1,5 nmol/l, although higher concentrations may be required and the value can vary between laboratories. The likelihood of toxicity rises dramatically at levels above 2 nmol/l.

The features of digoxin toxicity include:

- · Nausea and vomiting
- Diarrhoea
- · Abdominal pain
- Confusion
- · Tachyarrhythmias or bradyarrhythmias
- · Xanthopsia (yellow-green vision)
- · Hyperkalaemia (early sign of significant toxicity)

### Potential precipitating factors include:

- · Elderly patients
- · Renal failure
- Myocardial ischaemia
- Hypokalaemia
- Hypomagnesaemia
- Hypercalcaemia
- Hypernatraemia.
- Acidosis
- Hypothyroidism

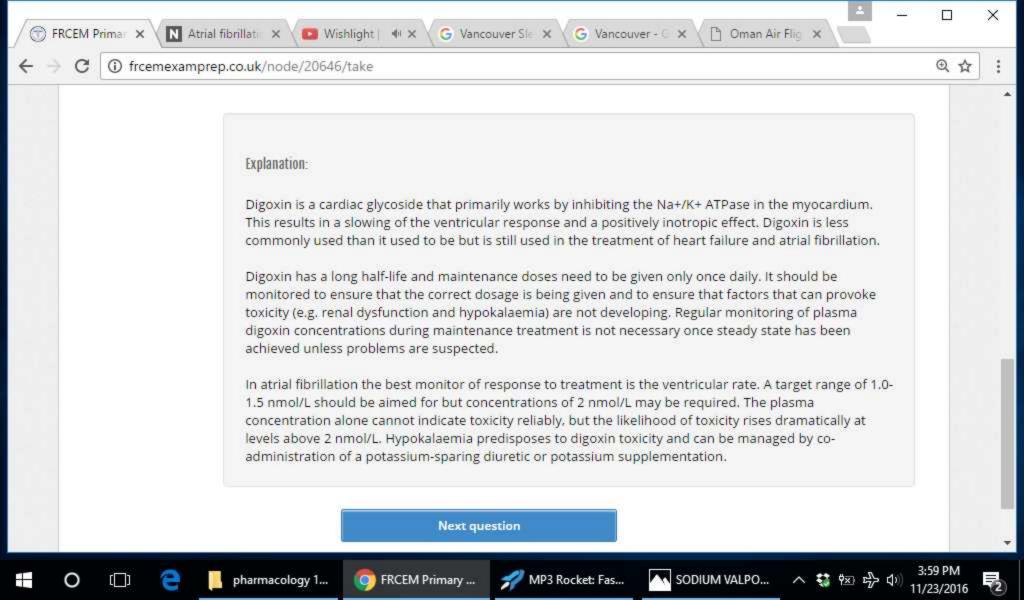
Numerous drugs can also predipose to digoxin toxicity including:

- Spironolactone
- Amiodarone
- Quinidine
- Verapamil
- Diltiazem
- · Drugs causing hypokalaemia e.g. thiazide and loop diuretics

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Pigoxin posture inotherise flowed excueted or 36 1 13 14 Manufu exceller 850 by aday). genal impaismet (many excepted 85%) by (colony) Amidorom UBB The Kuly Coul >2 or 200 novol

+ threpuli raise (1.3-2.6) toxity loud >2 o Reverse Tiche indicate Rigorin therapy · Degoxin toxicity may presented binition to Gastraeiditi -say>-65 e Digger SES xanthopsen underlyny Heart disease o disk Factor For digoxi's preexisting rever Failure ois wot First Doc in prement Amid Fibrilit (BY) o Can Canso Gryneconstre, Hass Darrow threputic window about Digbind => jummigletuli From Sty jumipile putigen burdy authorizing Should be used somer tact by Binding to digren millit Res - Bridy Action of YON



Distal convoluted tubule	4	

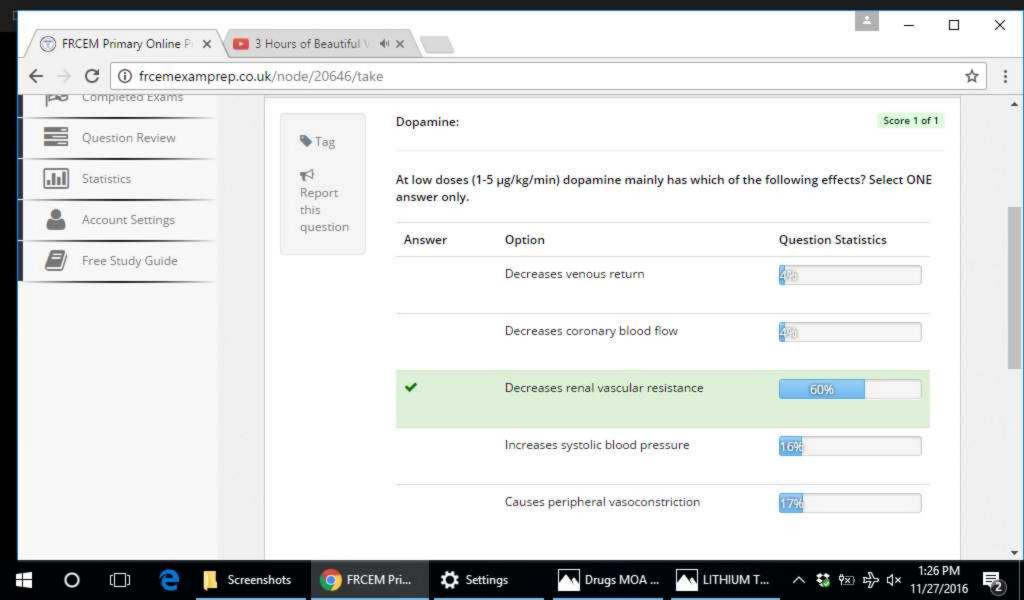
### Explanation:

Loop diuretics act on the Na.K.2CI co-transporter in the ascending loop of Henlé to inhibit sodium, chloride and potassium reabsorption. This prevents the generation of a hypertonic renal medulla and reduces the osmotic gradient that forces water to leave the collecting duct system. This has a powerful diuretic effect.

The following table summarises the mechanism of action of the different types of diuretic:

Descending loop of Henlé

Diuretic	Mechanism of action
Loop diuretics e.g. furosemide, bumetanide	Act on the Na.K.2Cl co-transporters in the ascending loop of Henlé to inhibit sodium, chloride and potassium reabsorption.
Thiazide diuretics e.g. bendroflumethiazide, hydrochlorothiazide	Act on the Na.Cl co-transporter in the distal convoluted tubule to inhibit sodium and chloride reabsorption.
Osmotic diuretics e.g. mannitol	Increases the osmolality of the glomerular filtrate and tubular fluid, increasing urinary volume by an osmotic effect.
Aldosterone antagonists e.g. spironolactone	Acts in the distal convoluted tubule as a competitive aldosterone antagonist resulting in inhibition of sodium reabsorption and increasing potassium reabsorption.
Carbonic anhydrase inhibitors e.g. acetazolamide	Inhibits the enzyme carbonic anhydrase preventing the conversion of bicarbonate and hydrogen ions into carbonic acid. This reduces the availability of hydrogen ions and causes sodium and bicarbonate to remain in the renal tubule resulting in diuresis.







Omeprazole 25%

Furosemide



# **Explanation:**

Drugs that cause gynaecomastia include:

- Cimetidine
- Omeprazole
- Spironolactone
- Digoxin
- Furosemide
- Finasteride
- Some anti-psychotics.

Ranitidine dose not tend to cause gynaecomastia and in fact gynaecomastia caused by cimetidine has been shown to resolve when it has been substituted with ranitidine.

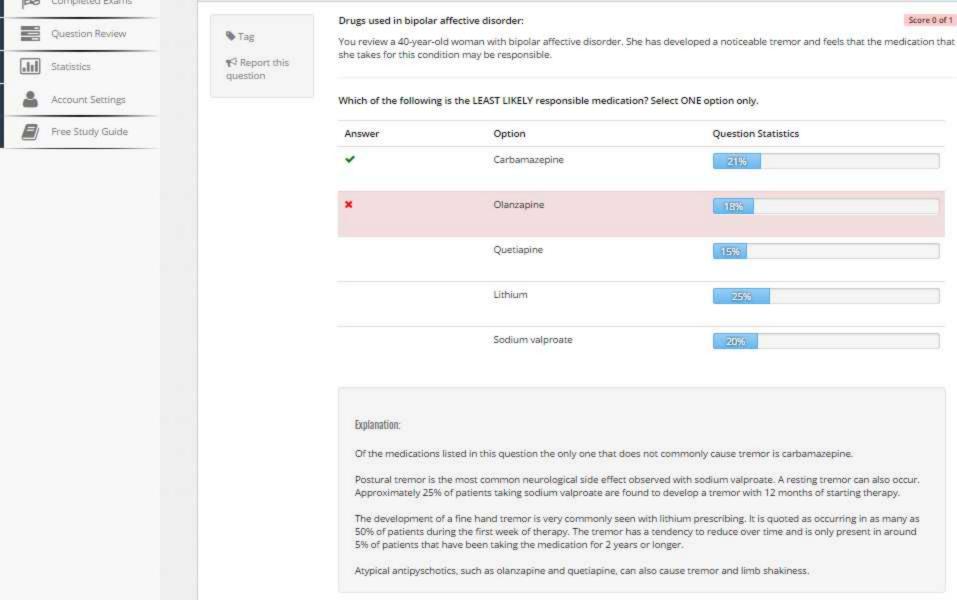
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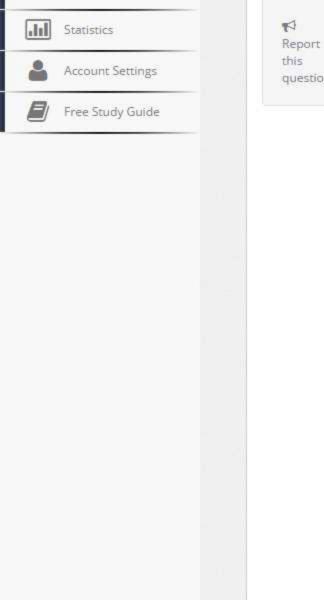








Score 0 of 1



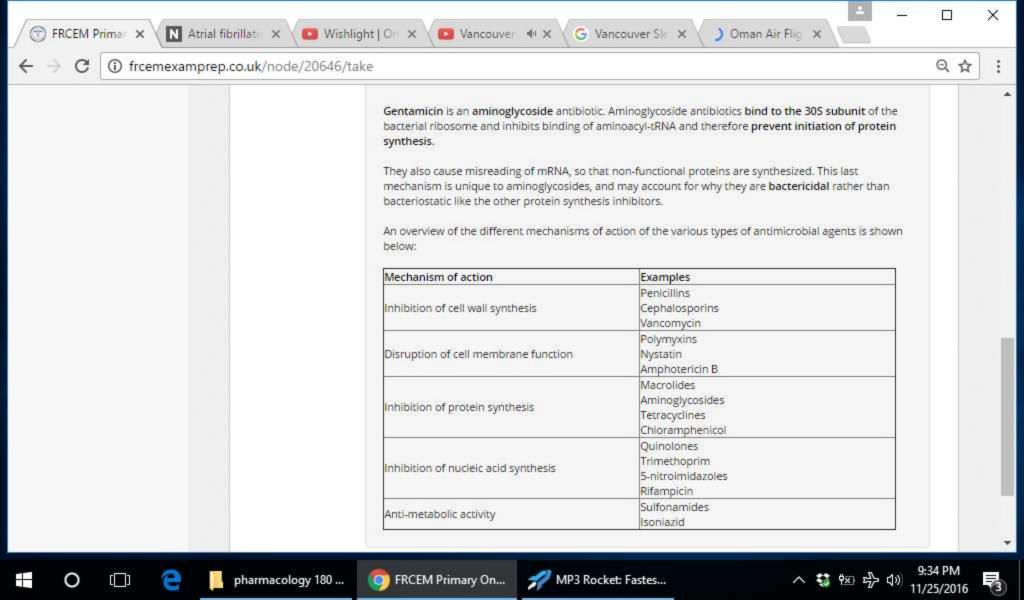
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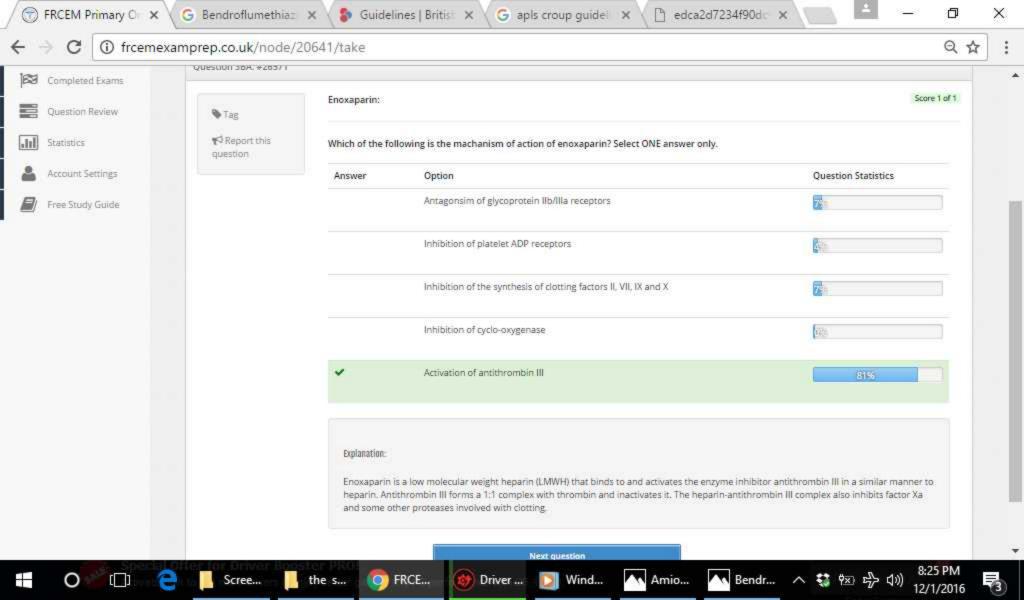
Which of the following drugs is NOT associated with possible hyperpigmentation? Select ONE answer only.

Answer	Option	Question Statistics
~	Azithromycin	5196
	Minocycline	9%
	Chloroquine	7
	Chlorpromazine	20%
	Amiodarone	13%
	Amiodarone	

# Explanation:

- Drugs that are commonly known to cause hyperpigmentation include: Chlorpromazine – grey pigmentation in sun-exposed parts of the body
- Psoralens
- . Minocycline blue/ black pigmentation of buccal mucosa and in scars
- Amiodarone blue/grey pigmentation occurring in sun-exposed parts of the body · Chloroquine - blue/grey pigmentation of face and arms





Explanation.

Entonox is a 50/50 mix of oxygen and nitrous oxide. Its main actions are analgesia and depression of the central nervous system. It is not known for certain how it works but it is postulated that it acts via the modulation of enkephalins and endorphins within the central nervous system.

Entonox takes approximately 30 seconds to act and continues for approximately 60 seconds after inhalation has ceased.

Entonox is stored in white or blue cylinders with blue and white shoulders. It has several uses including:

- As an adjuvant to general anaesthesia
- As an analgesic during labour
- As an analgesic during painful procedures

Recognized side effects of Entonox include:

- Nausea and vomiting (15% of patients)
- Dizziness
- Euphoria
- Inhibition of vitamin B12 synthesis

The following are contraindications to the use of entonox:

- · Reduced conscious level
- Diving injury
- Pneumothorax
- Middle ear disease
- Sinus disease
- Bowel obstruction
- Documented allergy to Entonox
- Hypoxia
- Violent / disabled psychiatric patients



Etomidate is a short acting carboxylated imidazole derivate that is primarily used for the induction of anaesthesia.

It is thought to act upon GABA type A receptors to modulate fast inhibitory synaptic transmission within the central nervous system.

The dose for induction of anaesthesia is 0.3 mg/kg. Following intravenous injection etomidate acts in 10-65 seconds and its duration of action is 6-8 minutes. Its effects are non-cumulative with repeated administration.

Etomidate is notable for its relative cardiovascular stability. It causes less hypotension than thiopental sodium and propofol during induction. It is also associated with rapid recovery without a hangover effect.

Etomidate is a potent inhibitor of steroidogenesis. Adrenal 11 beta-hydroxylase and cholesterol cleavage enzymes are inhibited by the drug, resulting in depression of cortisol and aldosterone synthesis for 24 hours after administration. Because of this adrenocortical suppression it should not be used for maintenance of anaesthesia.

Other adverse effects associated with the use of etomidate include:

- Nausea and vomiting
- Pain on injection (in up to 50%)
- Phlebitis and venous thrombosis
- Arrhythmias and heart block
- Hyperventilation
- · Respiratory depression and apnoea
- · Can cause both hypo- and hypertension
- · Increased mortality in critically ill patients



# 7:50 PM

**6** 52%

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Non-steroidal anti inflammatories (NSAIDs): Score 0 of 1

You review a patient with a knee injury and are considering prescribing him a non-steroidal anti inflammatory (NSAID) for pain relief.

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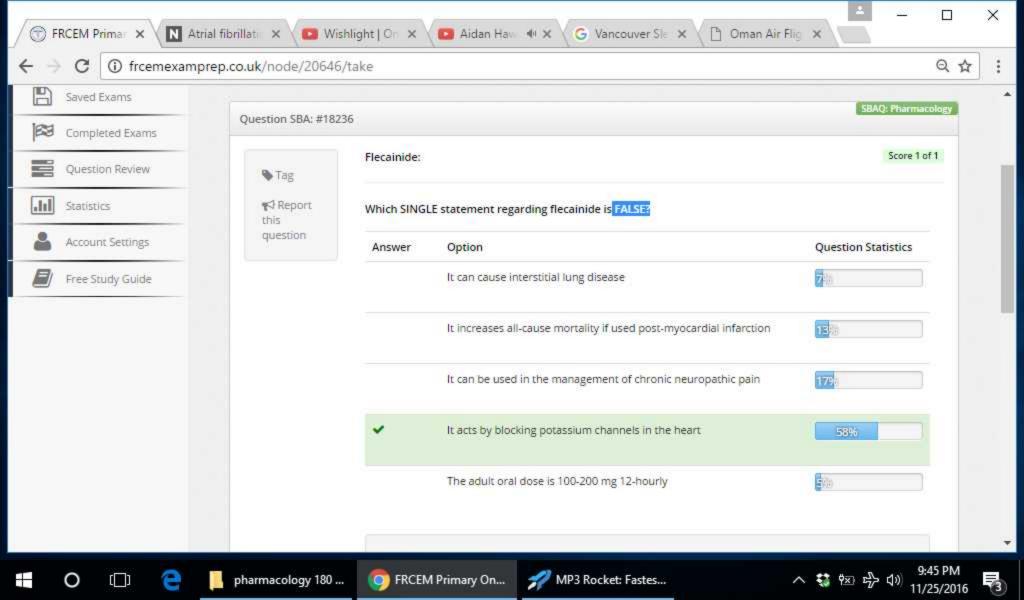
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Share...

Which of the following statements regarding NSAIDs is FALSE? Select ONE answer only.

Answer	Option	Question Statistics
~	Side effects are less commonly seen with indomethacin than naproxen	41%
×	It can take 21 days for full anti-inflammatory effect to become apparent	23%
	It can take 7 days for full analgesic effect to become apparent	15%
	Most NSAIDS act as non- selective inhibitors of the enzyme cyclo-oxygenase	9%
	Only approximately 60% of patients will respond	12%

to any given NSAID



Flecainide is a class ic antiarrhythmic agent that acts by blocking the Nav1.5 sodium channel in the heart, thereby prolonging the cardiac action potential and slowing conduction of the cardiac impulse within the heart. It has a profound effect on conduction in accessory pathways, especially on retrograde conduction, and markedly suppresses ventricular ectopic foci.

Flecainide can be used in the treatment of many different arrhythmias including:

- · Pre-excitation syndromes (e.g. Wolff-Parkinson-White)
- Acute atrial arrhythmias
- Ventricular arrhythmias

It has also been shown to be effective in the treatment of chronic neuropathic pain, www.ncbi.nlm.nih.gov.

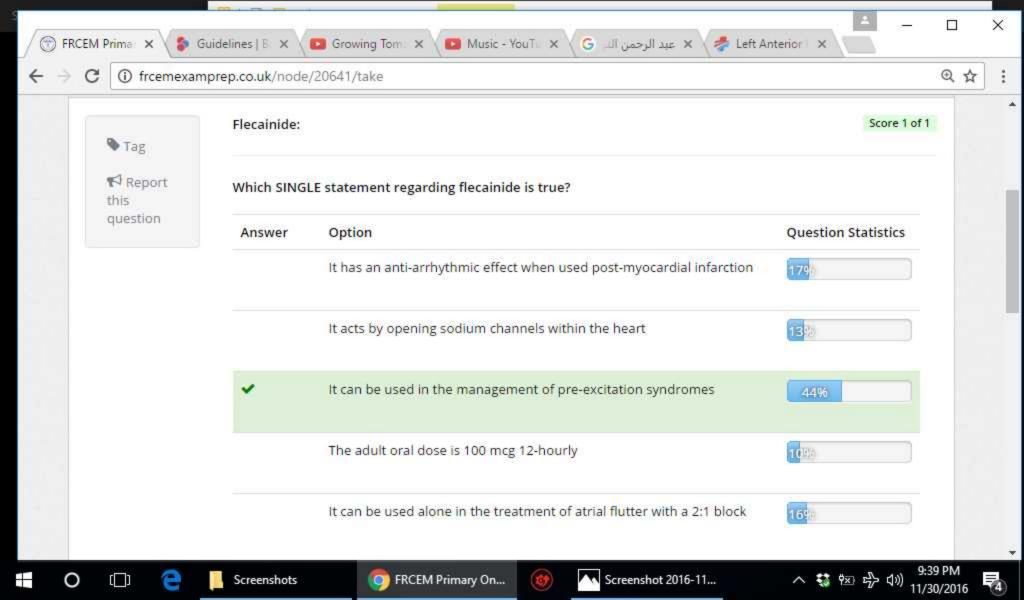
The adult oral dose is 100-200 mg 12-hourly. Intravenously it may be administered as a bolus dose of 2 mg/kg over 10 minutes followed b an infusion of 1.5 mg/kg/hour for one hour, reducing to 0.25 mg/kg/hour.

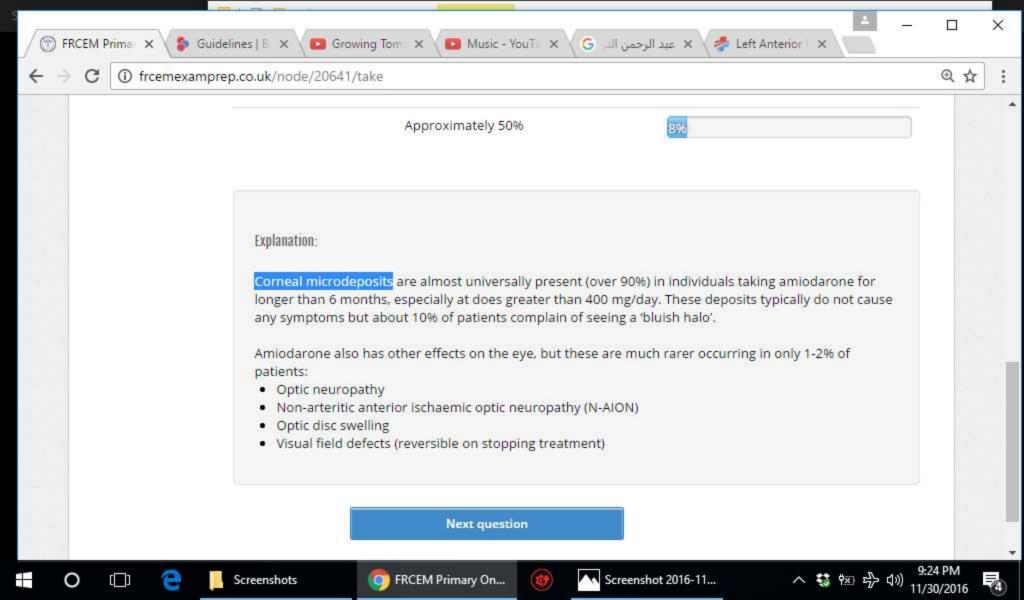
Flecainide should not be alone in the treatment of atrial flutter. If used alone there is a risk of inducing 1:1 atrioventricular conduction, with a consequent paradoxical increase in ventricular rate.

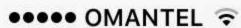
Flecainide is indicated only in patients without structural heart disease for the prevention, rapid control, or short-term prophylaxis of supraventricular and ventricular arrhythmias. The CAST trial showed a significant increase in sudden cardiac death and all-cause mortality in patients post-myocardial infarction, where is tended to be pro-arrhythmic, and in patients with an ejection fraction of < 40%. circ.ahajournals.org ©

Recognized side effects of flecainide include:

- Reversible liver toxicity
- Dizziness/vertigo
- Nausea and vomiting
- Visual disturbance
- Parasthesiae
- Interstitial lung disease



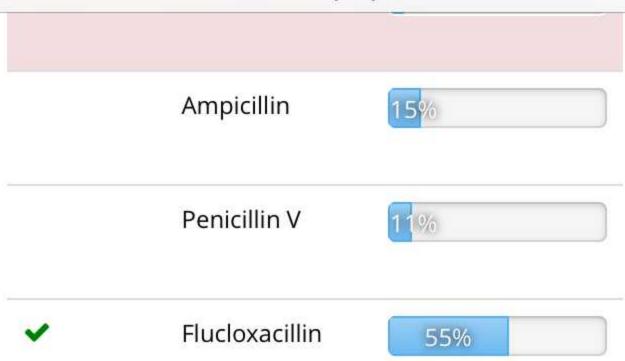




### 11:04 PM

19% [

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# **Explanation:**

The integrity of the **ß-lactam ring** is essential for antimicrobial activity. Many bacteria (including most staphylococci) are resistant to benzylpenicillin and phneoxymethylpenicillin because they produce enzymes (pencillinases, ß-lactamases) that open the ß-lactam ring.

Flucloxacillin is indicated in infections caused by penicillinase producing penicillin-resistant staphylococci. It is a semi-synthetic penicillin and is resistant to penicillinase because an isoxazolyl group sterically hinders access of the enzyme to the ß-lactam ring.

**Next question** 

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1:12 AM

⊕ 1 76% ■

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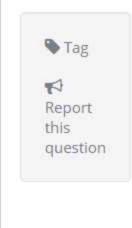
It can cause cardiac arrest in patient dependant on tricyclic antidepressants

# **Explanation:**

Flumazenil is a specific benzodiazepine antagonist that can be useful in some cases. It acts rapidly (in less than 1 minute) but the effects are short-lived and last less than 1 hour. The dose is 200 µg every 1-2 minutes (max dose 3mg / hour).

Flumazenil should be avoided if the patient dependant on benzodiazepine or takes tricyclic antidepressants as it can precipitate a withdrawal syndrome in these patients. In these circumstances it can cause seizures or cardiac arrest.

# **Next question**



Gastro-protection with NSAIDS:

Which of the following scenarios would NOT prompt you to consider the co-prescription of a PPI for gastro-protection with NSAIDs? Select ONE answer only.

Score 1 of 1

Answer	Option	Question Statistics
*	Long-term use for chronic back pain in a patient aged 30	30%
	Co-prescription of prednisolone	1096
	Co-prescription of fluoxetine	28%
	Short-term prescribing in a patient aged 67	24%
	Long-term use for rheumatoid arthritis in a patient aged 30	<b>7</b> 90

The current recommendations by NICE suggest that gastro-protection should be considered if patients have > 1 of the following:

- Using maximum recommended dose of an NSAID
- Aged 65 or older
- · History of peptic ulcer or GI bleeding
- Concomitant use of medications that increase risk
  - Low dose aspirin
  - Anticoagulants
  - Corticosteroids
  - Anti-depressants including SSRIs and SNRIs
- Requirements for prolonged NSAID usage
  - Patients with OA or RA at any age
  - Long-term back pain if older than 45

It is suggested that if required, either omeprazole 20 mg daily or lansoprazole 15-30 mg daily, should be the PPIs of choice.

This patient is on 400 mg of ibuprofen TDS but the maximum recommended dose of ibuprofen is 2.4 g daily. Co-prescription of codeine, raised BMI and a family history of peptic ulceration would also not prompt gastro-protection.

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Gentamicin is an aminoglycoside antibiotic. Aminoglycoside antibiotics bind to the 30S subunit of the bacterial ribosome and inhibits binding of aminoacyl-tRNA and therefore prevent initiation of protein synthesis.

They also cause misreading of mRNA, so that non-functional proteins are synthesized. This last mechanism is unique to aminoglycosides, and may account for why they are **bactericidal** rather than bacteriostatic like the other protein synthesis inhibitors.

Gentamicin is not absorbed orally and must be given by injection. It has a biological half-life of 2 hours and is renally excreted. Renal impairment results in accumulation and a greater risk of toxic side effects.

It is active against a wide range of Gramnegative and some Gram-positive organisms including:

- Pseudomonas spp.
- Escherichia coli
- Klebsiella pneumoniae
- Gram-positive Staphylococci spp.
- Yersinia pestis

Gentamicin should not be used for the treatment of infections caused by *Neisseria gonorrhoea*, *Neisseria meningitidis*, or *Legionella pneumophila* because of the risk of the patient going into shock from lipid A endotoxin release.

This patient has a classical presentation of temporal arteritis. Temporal arteritis, also known as giant cell arteritis (GCA), is a type of chronic vasculitis characterized by granulomatous inflammation in the walls of medium and large arteries. It usually affects people over 50 years of age.

#### Clinical features include:

- Headache
- Scalp tenderness
- Jaw claudication
- Amaurosis fugax or sudden blindness (typically unilateral).

Some patients also present with systemic features such as fever, fatigue, anorexia, weight loss, and depression.

It is associated with polymyalgia rheumatica (PMR) in 50% of cases (bilateral upper arm stiffness, aching, and tenderness; pelvic girdle pain).

Visual loss occurs early in the course of disease and, once established, it rarely improves.

Early treatment with high-dose corticosteroids is imperative to prevent further visual loss and other ischaemic complications. If GCA is suspected high-dose glucocorticosteroid treatment should be initiated immediately (40 - 60 mg prednisolone daily). An urgent referral for specialist evaluation (same day ophthalmology assessment for those with visual symptoms) and temporal artery biopsy should also be organised.

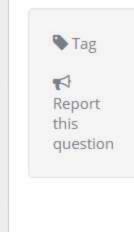
Hepatitis B vaccination isn't routinely available as part of the NHS vaccination schedule. It is only offered to those thought to be at increased risk of hepatitis B or its complications.

The Hepatitis B vaccine is a conjugate vaccine that contains a surface antigen of the hepatitis virus (HBsAg), on an aluminium adjuvant to increase immunogenicity. It is made via a recombinant DNA technique.

In adults and older children the preferred site of injection is the deltoid muscle. The anterolateral thigh is preferred in younger children. Gluteal injection is not recommended as reduced efficacy has been reported.

The standard regime is three primary doses (the initial dose, then further doses at one and six months later) with a booster at five years if still at risk. The accelerated regime for post-exposure prophylaxis is a vaccination at the time of exposure, then repeat doses at one and two months later.

Hepatitis B immunoglobulin can be given up to 7 days after high-risk exposure. Ideally immunoglobulin should be given within 12 hours but the BNF recommends use up to 7 days after exposure.



Hepatitis B prophylaxis:

You undertake a consultation to counsel a healthcare professional about hepatitis B prophylaxis.

Score 0 of 1

Which of the following statements regarding hepatitis B prophylaxis is true? Select ONE answer only.

Answer	Option	Question Statistics
×	Hepatitis B immunoglobulin can be given up to 28 days after high-risk exposure	10%
	The preferred site of injection is the gluteal area in adults	B9%
~	An accelerated regime is available for post-exposure prophylaxis	45%
	The hepatitis B vaccine is a live attenuated vaccine	946
	Hepatitis B vaccination is routinely offered as part of the NHS vaccination schedule	32%





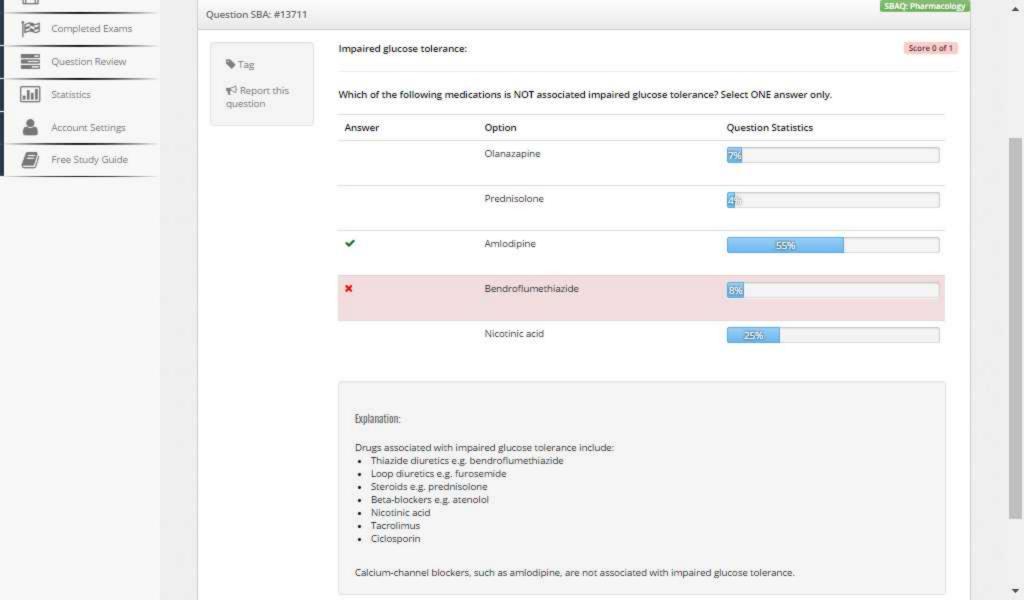
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Which of the following is the mechanism of action of heparin? Select ONE answer only.

Answer	Option	Question Statistics
	Inhibition of synthesis of factors I, III VII and VIII	10%
	Inhibition of synthesis of factors II, VII, IX and X	21%
	Inactivation of antithrombin II	10%
	Irreversible blockade of cyclo-oxygenase	3%
~	Activation of antithrombin III	55%

# **Explanation:**

Heparin binds to and activates the enzyme inhibitor antithrombin III. Antithrombin III forms a 1:1 complex with thrombin and inactivates it. The heparin-antithrombin III complex also inhibits factor Xa and some other proteases involved with clotting.

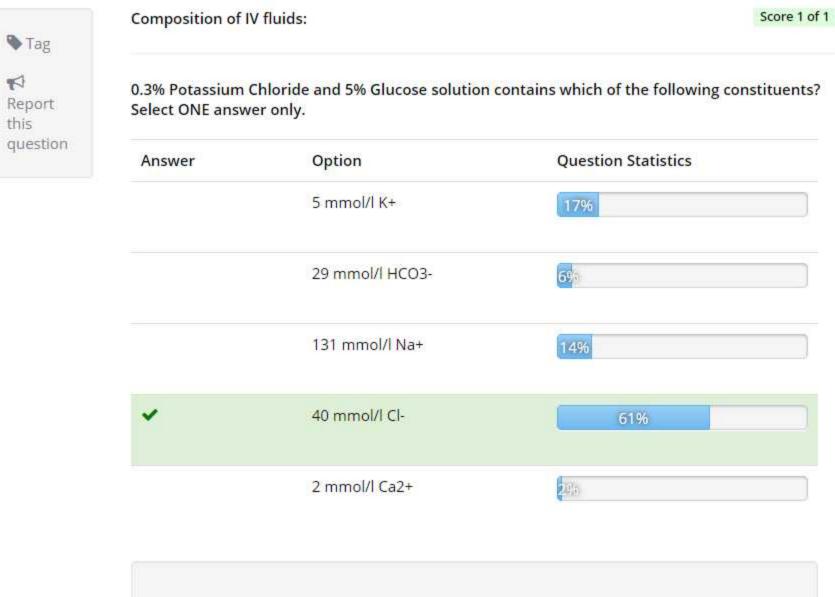




⊕ 53% □

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	псети	exampre	p.co.uk	E.	
FLUID	mmol/l	mmol/l	mmol/l	mmol/l	11111101/1
Normal					
plasma	142	4.5	26	103	2.5
values					
0.9%					
Sodium	150	-	=	150	-
Chloride					
Compound					
Sodium	121	_	20	111	2
Lactate	131	5	29	111	2
(Hartmann's)					
5% Glucose					
(1 L contains					
50 g of	-	-	-	13 <b>4</b>	-
dextrose)					
0.3%			3		
Potassium		40		40	
Chloride and	-	40	=	40	-
5% Glucose					
0.3%			-		
Potassium					
Chloride and	450	40		400	
0.9%	150	40	-	190	
Sodium					
Chloride					
1.26%			3		
Sodium	150	-	150	-	-
Bicarbonate					
4.5%			3		
Albumin (1 L					
contains 40-	< 160	< 2	=	136	-
50 g of					
albumin)					
4% Gelatin	154			120	.0.4
(Gelofusine)	154	< 0.4	-	120	< 0.4



The following table summarises the relative constituent compositions of the commonly used IV fluid mixtures (values taken from the BNF):

FLUID	Na+ mmol/l	K+ mmol/l	HCO <sub>3</sub> - mmol/l	CI- mmol/l	Ca <sup>2</sup> + mmol/l
Normal plasma values	142	4.5	26	103	2.5
0.9% Sodium Chloride	150	7.53	*	150	
Compound Sodium Lactate (Hartmann's)	131	5	29	111	2
5% Glucose (1 L contains 50 g of dextrose)		1.50	*	15	-
0.3% Potassium Chloride and 5% Glucose	-	40	-	40	-
0.3% Potassium Chloride and 0.9% Sodium Chloride	150	40	-	190	-
1.26% Sodium Bicarbonate	150		150	-	-
4.5% Albumin (1 L contains 40-50 g of albumin)	< 160	< 2	*	136	-
4% Gelatin (Gelofusine)	154	< 0.4		120	< 0.4



Ketamine is the only anaesthetic agent available that has analgesic, hypnotic, and amnesic properties. When used correctly it is a very useful and versatile drug.

Ketamine acts by non-competitive antagonism of the NMDA receptor Ca<sup>2+</sup> channel pore and also inhibits NMDA receptor activity by interaction with the phenylcyclidine binding site.

Ketamine can be used intravenously and intramuscularly. The intramuscular dose is 10 mg/kg and when used by this route it acts within 2-8 minutes and has a duration of action of 10-20 minutes. The intravenous dose is 1.5-2 mg/kg administered over a period of 60 seconds. When used intravenously it acts within 30 seconds and has a duration of action of 5-10 minutes. Ketamine is also effective when administered orally, rectally, and nasally.

Ketamine causes tachycardia, an increase in blood pressure, central venous pressure and cardiac output, secondary to an increase in sympathetic tone. Baroreceptor function is well maintained and arrhythmias are uncommon.

The main disadvantage to the use of ketamine is the high incidence of hallucinations, nightmares, and other transient psychotic effects. These can be reduced by the co-administration of a benzodiazepine, such as diazepam or midazolam.

The main side effects of ketamine are:

- Nausea and vomiting
- Hypertension
- Nystagmus
- Diplopia
- Rash

Lidocaine is a tertiary amine that is primarily used as a local anaesthetic but can also be used in the treatment of ventricular dysrrhythmias.

Lidocaine works as a local anaesthetic by diffusing in its uncharged base form through neural sheaths and the axonal membrane to the internal surface of the cell membrane sodium channels. Here it alters signal conduction by blocking the fast voltage-gated sodium channels. With sufficient blockage, the membrane of the postsynaptic neuron will not depolarize and will be unable to transmit an action potential, thereby preventing transmission of pain signals.

Each 1 ml of plain 1% lidocaine solution contains 10 mg of lidocaine hydrochloride. The maximum safe dose of plain lidocaine is 3 mg/kg. When administered with adrenaline 1:200,000 the maximum safe dose is 7 mg/kg. Because of the risk of vasoconstriction and tissue necrosis, lidocaine should not be used in combination with adrenaline in extremities such as fingers, toes, and the nose.

The half-life of lidocaine is 1.5-2 hours. Its onset of action is rapid within a few minutes and it has a duration of action of 30-60 minutes when used alone. Its duration of action is prolonged by coadministration with adrenaline.

Lidocaine tends to cause vasodilatation. This is believed to be due mainly to the inhibition of action potentials via sodium channel blocking in vasoconstrictor sympathetic nerves.

●●●●● OMANTEL 🤝 VPN

#### 7:19 PM

⊕ 60% □

### frcemexamprep.co.uk

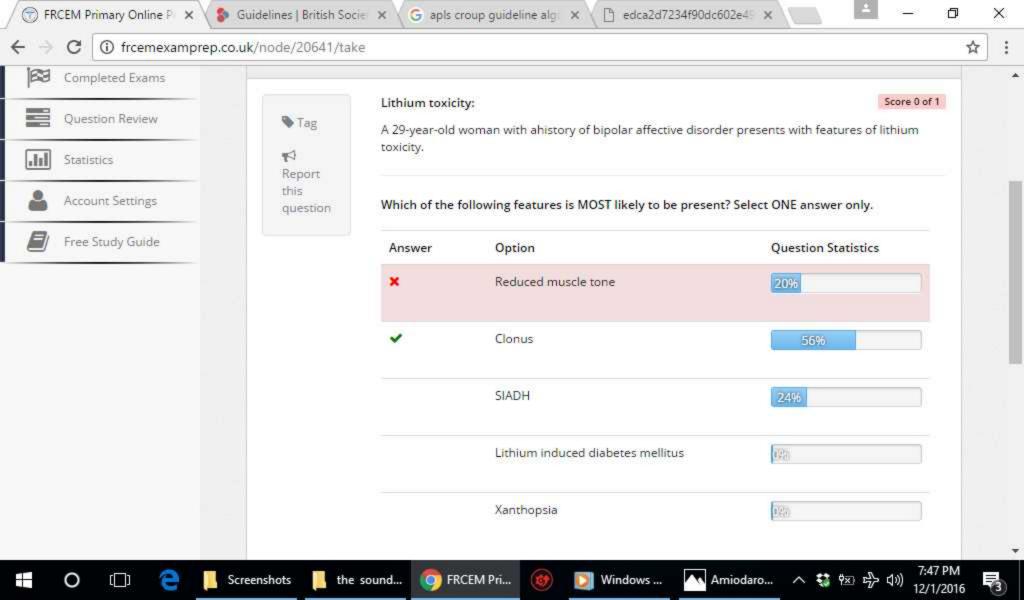
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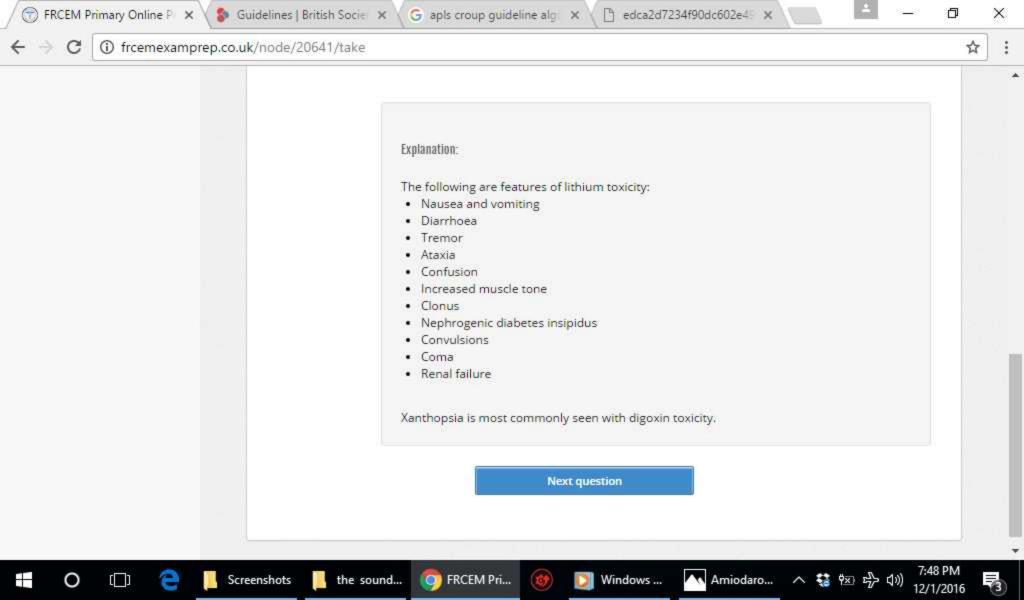
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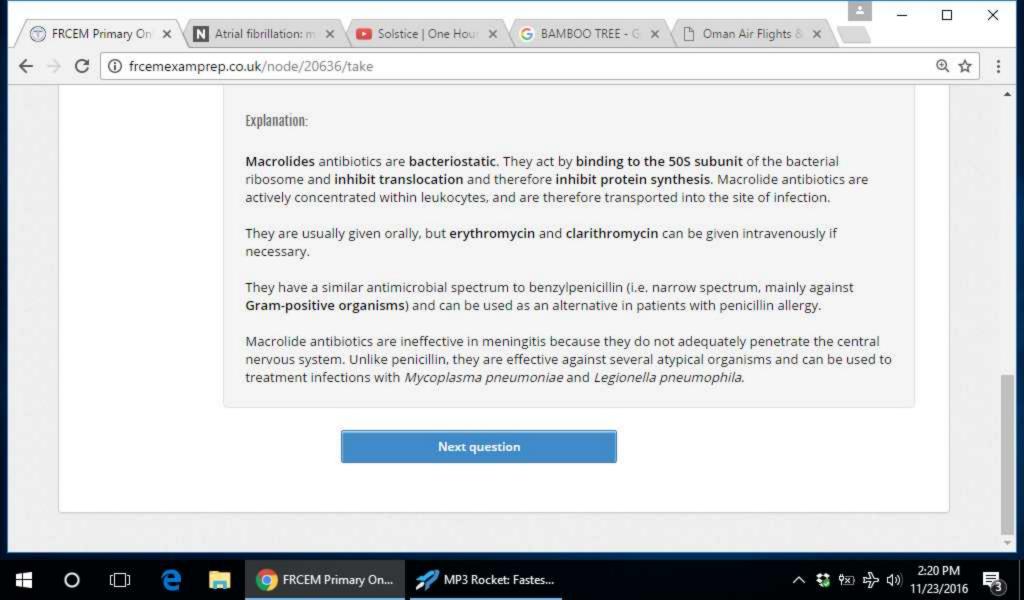
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# **Explanation:**

Metronidazole and the other 5-nitoimidazole agents inhibit nucleic acid synthesis by disrupting the DNA of microbial cells.

An overview of the different mechanisms of action of the various types of antimicrobial agents is shown below:

Mechanism of action	Examples
Inhibition of cell wall	Penicillins
	Cephalosporins
synthesis	Vancomycin
Disruption of cell	Polymyxins
membrane function	Nystatin
membrane function	Amphotericin B
	Macrolides
Inhibition of protein	Aminoglycosides
synthesis	Tetracyclines
	Chloramphenicol
	Quinolones
Inhibition of nucleic	Trimethoprim
acid synthesis	5-nitroimidazoles
	Rifampicin
Anti-metabolic	Sulfonamides
activity	Isoniazid

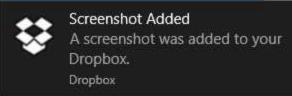
**Next question** 

NSAIDs are associated with a relatively high incidence of renal adverse drug reactions (ADRs). The principal mechanism by which these renal ADRs occur is due to changes in renal heamodynamics via changes in prostaglandin levels.

Prostaglandins normally cause vasodilatation of the afferent arteriole of the glomerulus, which preserves normal glomerular perfusion and glomerular filtration rate (GFR).

NSAIDs reduce prostaglandin levels, which causes unopposed vasoconstriction of the afferent arteriole and decreased renal plasma flow, this in turn causes a decreased GFR. NSAIDs have no effect, however, on the filtration fraction itself.

**Next question** 



2/10/6

# Which of the following statements regarding NSAIDs is true? Select ONE answer only.

Answer	Option	Question Statistics
	Co-prescribing a proton pump inhibitor would be unusual	59/6
~	Regular use of aspirin is associated with around a 20% increase in erectile dysfunction	26%
	Naproxen interacts with aspirin by antagonizing the irreversible platelet inhibition	21%i
	Diclofenac is safe to prescribe in a patient that has had an MI a year ago	25%

NSAIDs and COX-2 inhibitors vary greatly in their analgesic efficacy

NSAIDs and COX-2 inhibitors are very similar in their analgesic properties, but vary more widely in their side-effect profiles.

NICE recommends adding in a proton pump inhibitor when prescribing NSAIDs.

All NSAIDs have been linked with an increased risk of death or recurrent MI and thus their prescribing should be very carefully considered in patients with a significant cardiac history.

Naproxen appears to be the safest NSAID with regards to cardiovascular side-effects, and it doesn't interact with aspirin.

Regular use of aspirin is associated with around a 20% increase in erectile due function



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Dropbox



### 7:51 PM

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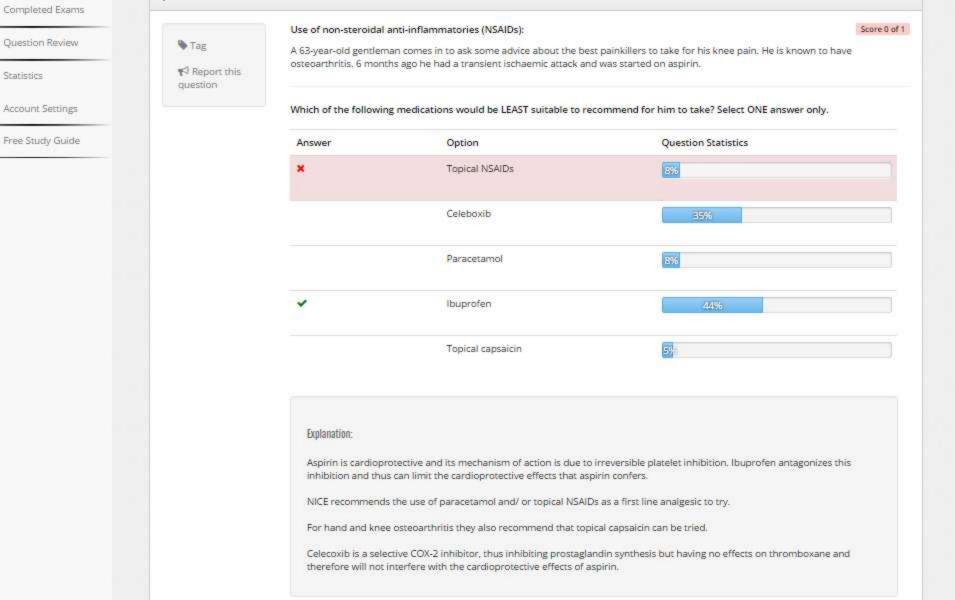
# **Explanation:**

Most NSAIDS act as non-selective inhibitors of the enzyme cyclo-oxygenase (COX), inhibiting both the COX-1 and COX-2 isoenzymes. COX catalyses the formation of prostaglandins and thromboxane from arachidonic acid. These in turn act as messenger molecules in the process of inflammation.

There is also considerable variance in how well the various NSAIDs are tolerated, but in generally side effects are lowest with Ibuprofen, then naproxen, then diclofenac then indomethacin. Therefore side effects are more commonly seen with indomethacin than naproxen.

Pain relief starts soon after the first dose but it can take a week to reach full analgesic effect. Only approximately 60% of patients will respond to any given NSAID. A clinically appreciable reduction in inflammation may not be apparent until 21 days of treatment. If no improvement is seen by 21 days, a different NSAID should be trialled.

**Finish** 



dil



### 7:50 PM

**6** 52%

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Non-steroidal anti inflammatories (NSAIDs): Score 0 of 1

You review a patient with a knee injury and are considering prescribing him a non-steroidal anti inflammatory (NSAID) for pain relief.

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Which of the following statements regarding NSAIDs is FALSE? Select ONE answer only.

Answer	Option	Question Statistics
~	Side effects are less commonly seen with indomethacin than naproxen	41%
×	It can take 21 days for full anti-inflammatory effect to become apparent	23%
	It can take 7 days for full analgesic effect to become apparent	15%
	Most NSAIDS act as non- selective inhibitors of the enzyme cyclo-oxygenase	9%
	Only approximately 60% of patients will respond	12%

to any given NSAID

expialiation.

Opioid poisoning is a relatively common Emergency Department presentation. Overdose can be secondary to recreational drug (e.g. heroin) or as a consequence of prescribed opioids (e.g. morphine sulfate tablets, dihydrocodeine).

The clinical features of opioid overdose include:

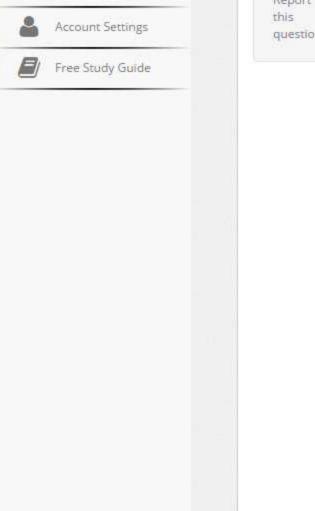
- Reduced conscious level or coma
- · Reduced respiratory rate
- Apnoea
- Pinpoint pupils
- Hypotension
- Cyanosis
- Convulsions
- Non-cardiogenic pulmonary oedema (with IV heroin usage)

The main cause of death secondary to opioid overdose is respiratory depression, which usually occurs within 1 hour of the overdose. Vomiting is also common and aspiration can occur.

Naloxone is the specific antidote for opioid overdose and will reverse respiratory depression and coma if given at sufficient dosage. The initial dose is usually 0.8 mg (2 mL) intravenously (the dose range suggested by BNF is 0.4-2 mg). It can also be given by intramuscular injection if the intravenous route is not feasible.

As naloxone has a shorter duration of action than most opioids, close monitoring and repeated injections are necessary according to the respiratory rate and depth of coma. The dose is generally repeated every 2-3 minutes to a maximum of 10 mg. When repeated doses are needed naloxone may be given by a continuous infusion adjusted according to the vital signs. Initially the infusion rate can be set at 60% of the initial resuscitative IV dose per hour.

In opioid addicts naloxone administration may precipitate a withdrawal syndrome with abdominal cramps, nausea and diarrhoea, but these usually settle within 2 hours.



this question

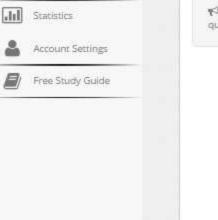
Which of the following can be used as an antidote for paracetamol overdose? Select ONE answer only.

Answer	Option	Question Statistics
	Glucagon	9%
	Desferrioxamine	6.
	Octreotide	448
*	Methionine	74%
	Fomepizole	65

Explanation:

The mainstay of treatment of paracetamol overdose is acetylcysteine. Acetylcysteine is a very effective antidote but its effectiveness declines rapidly if started > 8 hours after a significant ingestion. All ingestions > 75 mg/kg are considered to be significant.

Methionine is a useful alternative in patients who refuse treatment. It is given orally 2.5 g every 4 hours to a total dose of 10 g.



₹<sup>3</sup> Report this question

#### Which ONE of the following drugs is most likely to cause peripheral neuropathy as a side effect?

Answer	Option	Question Statistics
	Bisoprolof	
	Amiodarone	17%
~	Isoniazid	76%
	Dexamethasone	<b>3</b> 6
	Amlodipine	

#### Explanation:

Isoniazid is a first-line agent for the treatment of tuberculosis. One of the commonest side effects of isoniazid is peripheral neuropathy, which occurs in up to 20% of patients taking the drug at a dose of greater than 6 mg/kg daily.

Peripheral neuropathy occurs because isoniazid combines with pyridoxine (vitamin B6) to form hydrazone, which is subsequently excreted in the urine. This results in a deficiency of biologically active pyridoxine that results in a peripheral neuropathy.

The peripheral neuropathy of isoniazid can be prevented by the co-administration of pyridoxine at a dose of 10 mg for each 100 mg of isoniazid given. The administration of pyridoxine does not interfere with the antituberculous action of isoniazid.

Next question

•••• OMANTEL 3G

1:24 AM

⊕ 76% ■

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wnich of the following vaccinations are safe to administer to a pregnant patient? Select ONE option only.

Answer	Option	Question Statistics
	MMR vaccine	15%
	Anthrax vaccine	6%
	HPV vaccine	13%
	Varicella vaccine	24%
~	Pertussis vaccine	42%

# **Explanation:**

Pertussis vaccination is now recommended for pregnant patients due to the high complication rates of whooping cough in pregnancy.

The others are not recommended when pregnant and for full guidelines please go to:

www.cdc.gov



### 7:06 PM

⊕ 64% 
□

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Prescribing in epilepsy:

Score 1 of 1

A 38-year-old female patient with epilepsy complains of feeling depressed. You notice that she has coarse facial features, gum hypertrophy and prominent facial acne. She also has an ataxic gait when she walks.

Which of the following anti-epileptic medications is most likely to be responsible for her presentation? Select ONE answer only.

Answer	Option	Question Statistics
	Levetiracetam	2%
	Carbamazepine	8%
•	Phenytoin	72%
	Vigabatrin	2%
	Sodium valproate	16%

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Amiodarone has many potential toxic side effects and a full and thorough clinical assessment prior to commencing therapy with it is essential.

Side effects associated with amiodarone include:

- Corneal microdeposits
- Photosensitivity
- Nausea
- Sleep disturbance
- Hyperthyroidism
- Hypothyroidism
- · Acute hepatitis and jaundice
- · Peripheral neuropathy
- · Lung fibrosis
- QT prolongation

Optic neuritis is a very rare side effect of amiodarone. If it does occur then the amiodarone should be stopped immediately due to the risk of blindness.

Most patients taking amiodarone develop corneal microdeposits, this reverses after treatment has been ceased and rarely interferes with vision.

Amiodarone chemically resembles thyroxine and can bind to the nuclear thyroid receptor. It can cause both hypothyoidism and hyperthyroidism, although hypothyroidism is far more common, occurring in 5-10% of patients.

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Report this question

# Side effects of tetracyclines:

Score 1 of 1

A 26-year-old man develops a severe skin rash 2 weeks after being prescribed doxycycline. He initially felt generally unwell with a mild fever and flu-like symptoms. He subsequently developed a rash, which started as numerous 'target lesions' and has now progressed to severe bullous, ulcerating skin lesions with areas of epidermal detachment. You estimate that the epidermal detachment is affecting 8% of his total body surface area. He has severe mouth and tongue ulceration, which is shown in the photo below.



●●●● OMANTEL 🖘

## 11:46 AM

⊕ 51%

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Explanation:

Stevens-Johnson syndrome is a severe and potentially fatal form of erythema multiforme. It can be caused by anything that causes erythema multiforme but is most frequently seen as a drug reaction 1-3 weeks after initiation. There is often an initial prodrome with constitutional symptoms such as fever, malaise, arthralgia and gastrointestinal upset followed by the appearance of severe mucocutaneous lesions, which are bullous and ulcerating.

Stevens-Johnson syndrome and toxic epidermal necrolysis are considered to be a single mucocutaneous disease with an increasing severity. They can be differentiated by the degree of epidermal detachment seen. In Stevens-Johnson syndrome epidermal detachment is seen in less than 10% of the body surface area, whereas in toxic epidermal necrolysis epidermal detachment is seen in greater than 30% of the body surface area. An overlap syndrome exists when detachment is between 10-30% of the body surface area

Drugs that can cause Stevens-Johnson syndrome and toxic epidermal necrolysis include:

- Tetracyclines
- Penicillins
- Vancomycin
- Sulphonamides
- NSAIDs
- Barbiturates

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# **Explanation:**

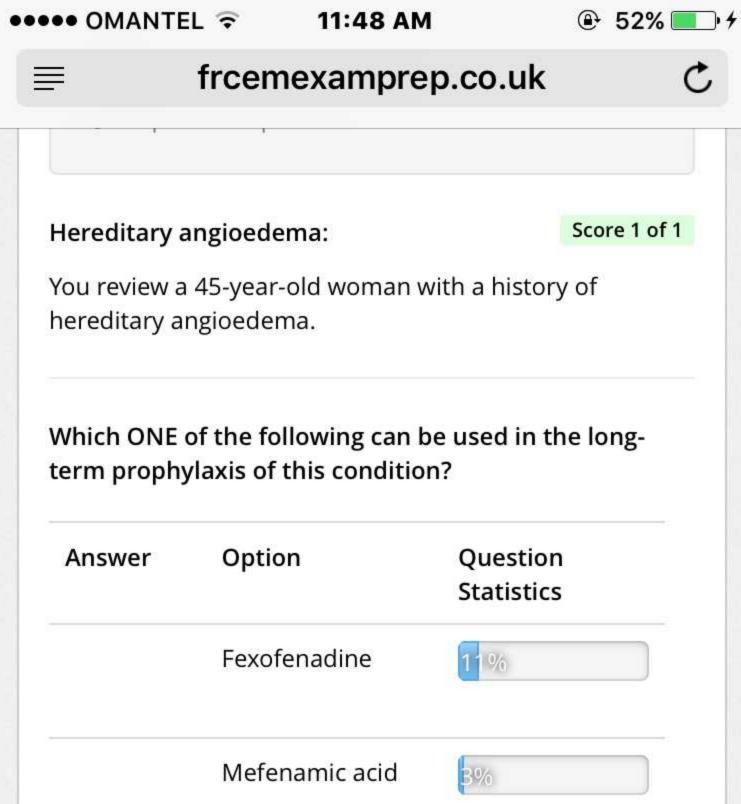
Hereditary angioedema is caused by a deficiency of C1 esterase inhibitor, a protein that forms part of the complement system. It is usually inherited in an autosomal dominant fashion.

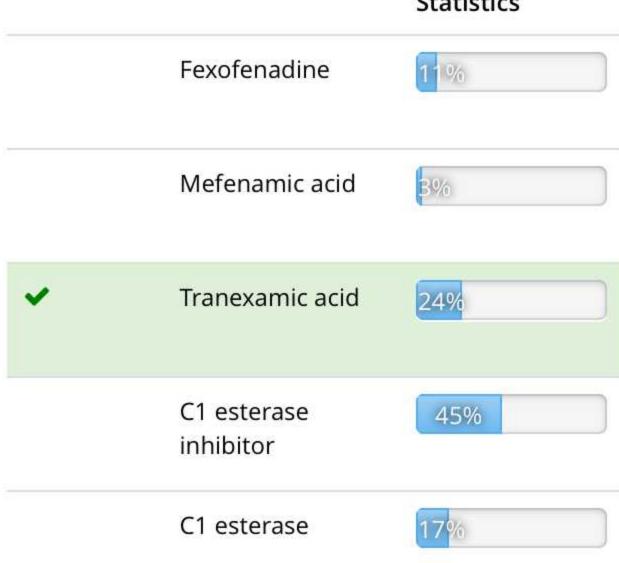
Symptoms usually begin in childhood and occur sporadically throughout adult life. Attacks can be precipitated by minor surgical and dental procedures and stress. The main clinical features of hereditary andioedema are oedema of the skin and mucous membranes. The most commonly affected areas are the face, tongue and extremities. There is often a prodrome of tingling and it is sometimes preceded by a nonpruritic rash.

Angioedema and anaphylaxis due to C1 esterase inhibitor deficiency is resistant to adrenaline, steroids and antihistamines and needs treatment with C1 esterase inhibitor concentrate or fresh frozen plasma, which contains C1 esterase inhibitor.

Short-term prophylaxis for situations that may precipitate an attack can be achieved with C1 esterase inhibitor or fresh frozen plasma infusions prior to the event.

Long-term prophylaxis can be achieved with androgenic steroids such as stanozolol or antifibrinolytic drugs such as tranexamic acid.











Score 0 of 1



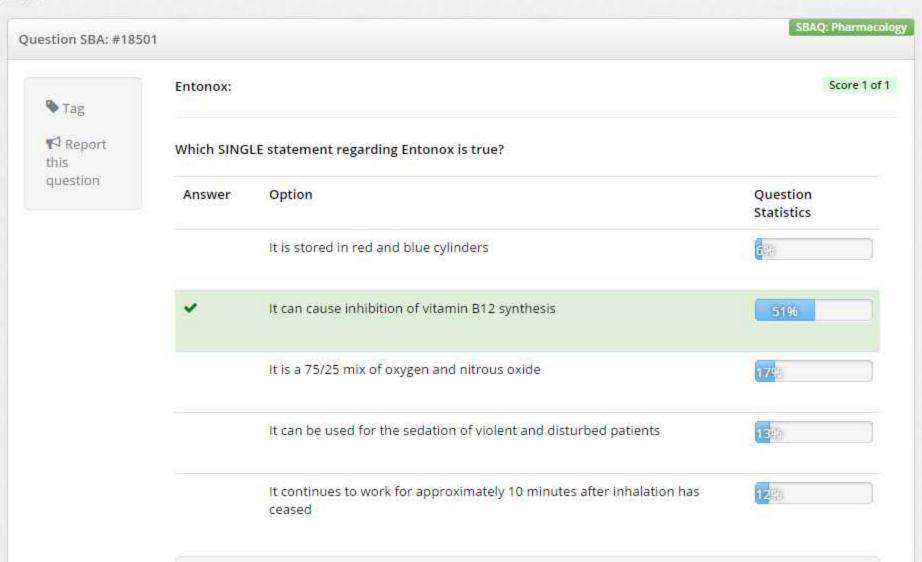
Report this question

## Proton pump inhibitors:

You see a 42-year-old women with epigastric pain. Her only medication is omeprazole, which she has been taking for 6 months.

## Which SINGLE statement regarding proton pump inhibitors is true?

Answer	Option	Question Statistics
~	They are associated with a risk of low serum magnesium levels	46%
	They are effective in the treatment of gastric ulcers but not duodenal ulcers	EU.
	They are associated with an increased risk of pelvic fracture	15%
×	IV PPIs should be given for peptic ulcer bleeding in preparation for endoscopy	3196
	The proton pump is the first stage in gastric acid secretion	493



Proton pump inhibitors act by blocking the hydrogen/potassium ATPase enzyme system of the gastric parietal cells. The proton pump is the terminal stage in gastric acid secretion and this makes the proton pump an ideal target for inhibiting acid secretion.

The outcome is similar with both oral and intravenous PPI use and there is no appreciable benefit for using the intravenous formulation in patients that can tolerate oral medication.

Long-term PPI use has been associated with an increased risk of hip, wrist and spine fractures, but not pelvic fractures.

There is an increased risk of both *Clostridium Difficile* infection and community-acquired pneumonia with PPI usage. It is suspected that acid suppression caused by PPI usage results in poor elimination of pathogenic organisms leading to increased infection risk.

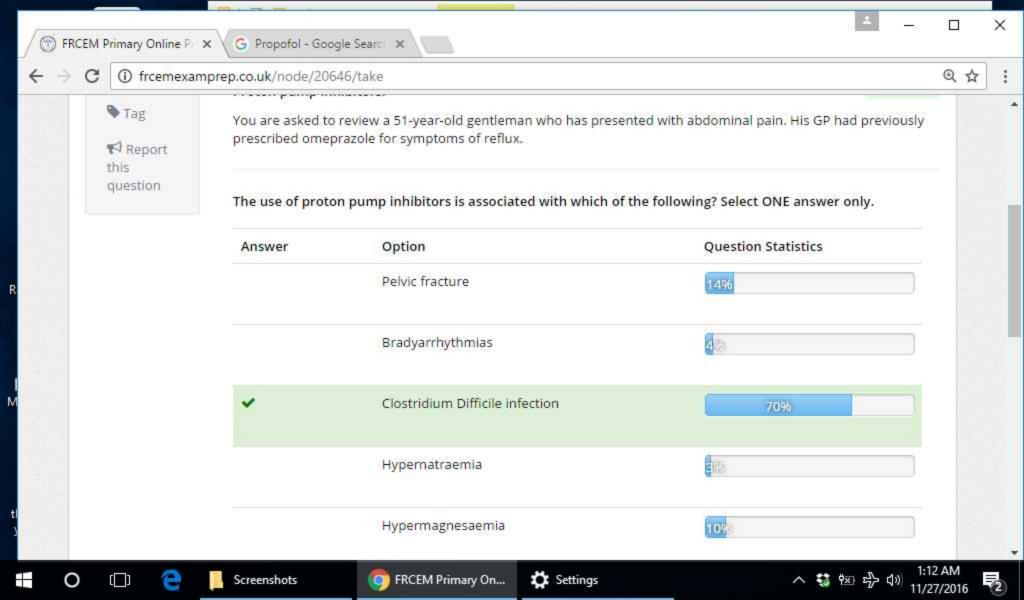
The current recommendations by NICE suggest that gastro-protection should be considered if patients have > 1 of the following:

- Using maximum recommended dose of an NSAID
- Aged 65 or older
- History of peptic ulcer or GI bleeding
- · Concomitant use of medications that increase risk
  - Low dose aspirin
  - Anticoagulants
  - Corticosteroids
  - Anti-depressants including SSRIs and SNRIs
- Requirements for prolonged NSAID usage
  - Patients with OA or RA at any age
  - Long-term back pain if older than 45

It is suggested that if required, either omeprazole 20 mg daily or lansoprazole 15-30 mg daily, should be the PPIs of choice.

A patient on 400 mg of ibuprofen TDS (the maximum recommended dose of ibuprofen is 2.4 g daily), co-prescription of codeine, raised BMI and a family history of peptic ulceration would all not prompt gastro-protection.

A useful Clinical Knowledge Summary by NICE on this topic can be viewed here: cks.nice.org.uk@



Proton pump inhibitors inhibit gastric acid secretion by blocking the hydrogen-potassium ATP enzyme system of the gastric parietal cell (the 'proton pump'). The proton pump is the terminal stage in gastric acid secretion and this makes it an ideal target for inhibiting acid secretion.

They are effective short-term treatments for both gastric and duodenal ulcers, and are also used in combination with antibacterials for the eradication of Helicobacter pylori. They can also be used for the treatment of dyspepsia and gastro-oesophageal reflux disease (GORD), and for the prevention and treatment of NSAID-associated ulcers.

Following endoscopic treatment of severe peptic ulcer bleeding, high-dose PPI therapy reduces risk of rebleeding and surgery. There is, however, no difference in outcome between oral and IV usage prior to endoscopy. The current SIGN guidelines recommend that PPIs should not be used prior to endoscopic therapy when early endoscopic examination is performed within 24 hours of admission.

Common side effects of PPIs include:

- Nausea and vomiting
- Abdominal pain
- Flatulence
- Diarrhoea
- Constipation
- Headache

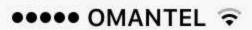
They are associated with an increased risk of Clostridium Difficile infection: http://www.ncbi.nlm.nih.gov/pubmed/25116712 ₪

There is also epidemiological evidence of increased fracture risk with long-term PPI usage. Observational studies have shown a modest association with hip, wrist and spine fractures. There is, however, no associated increase risk of pelvic fracture. The MHR advises that patients at risk of osteoporosis that take PPIs maintain an adequate intake of calcium and vitamin D for this reason.

PPIs have been shown to be associated with a significant risk of focal tachyarrhythmias: PPI-associated-risk-focal-arrhythmias  $\oplus$ 

The US FDA have highlighted a risk of low serum magnesium and low sodium levels in patients taking PPIs long-term; http://www.fda.gov/Drugs/DrugSafety/ucm245011.htm

€



# 11:13 PM

17% 💷

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Propofol:

Score 0 of 1

Which SINGLE statement regarding propofol is true?

Answer	Option	Question Statistics
×	It is thought to work by inhibiting GABA and glycine	19%
	It increases systemic vascular resistance	3%
~	It decreases cardiac output by approximately 20%	51%
	75% of patients experience pain on injection	21%
	It has positively inotropic effects	5%

**Explanation:** 

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# **Explanation:**

Propofol (2,6-diisopropylphenol) is a short acting phenol derivative that is primarily used for the induction of anaesthesia.

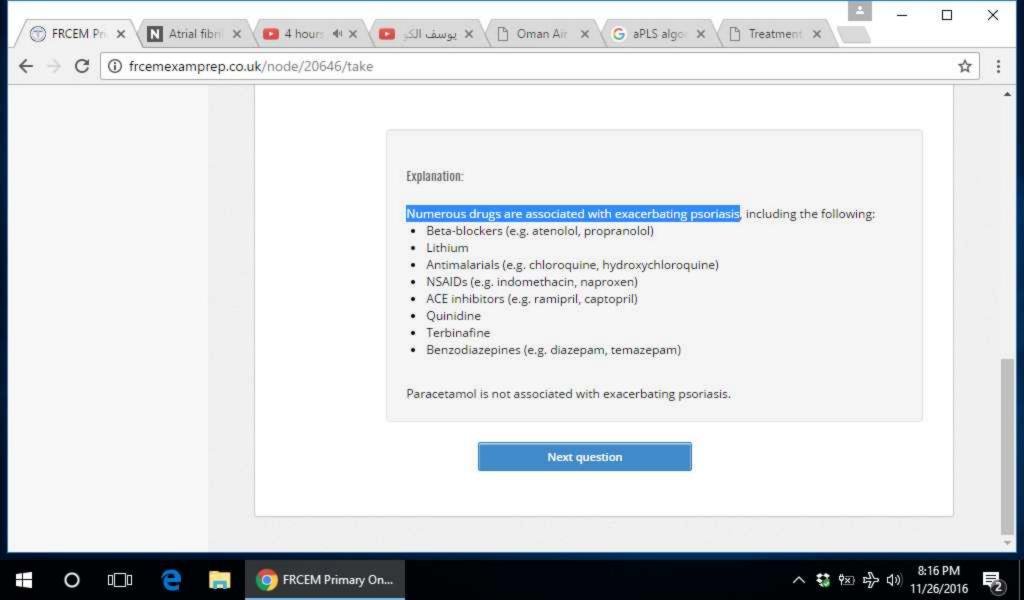
Its mechanism of action is unclear but is thought to act by potentiating the inhibitory neurotransmitters GABA and glycine, which enhances spinal inhibition during anaesthesia.

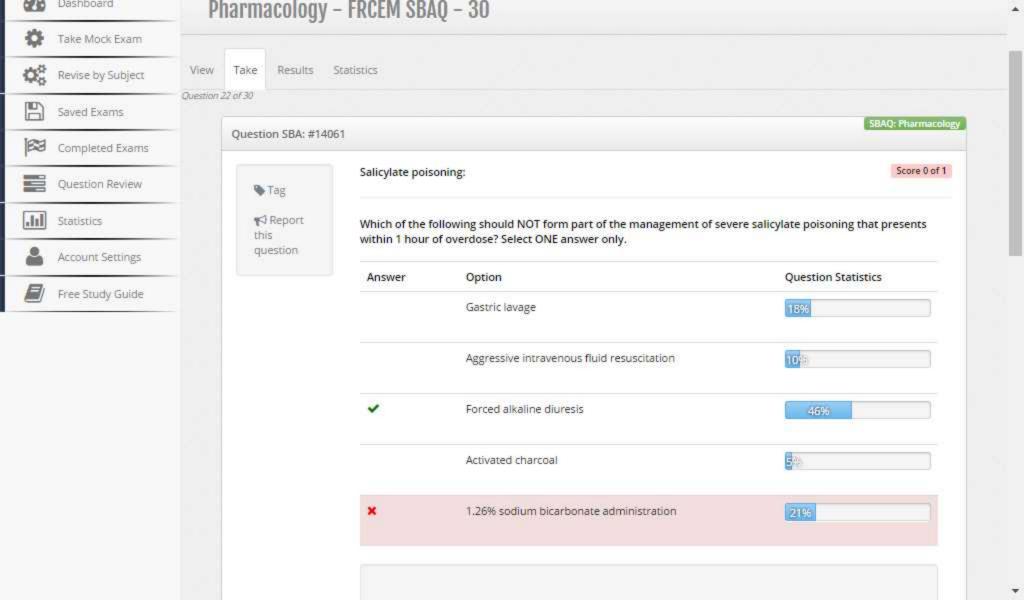
The dose for induction of anaesthesia is 1.5-2.5mg/kg. The dose for maintenance of anaesthesia is 4-12 mg/kg/hour. Following intravenous injection propofol acts within 30 seconds and its duration of action is 5-10 minutes.

Propofol produces a 15-25% decrease in blood pressure and systemic vascular resistance without a compensatory increase in heart rate. It is negatively inotropic and decreases cardiac output by approximately 20%.

The main side effects of propofol are:

- Pain on injection (in up to 30%)
- Hypotension
- Transient apnoea
- Hyperventilation
- Coughing and hiccough
- Headache
- · Thrombosis and phlebitis





 Nausea and vomiting Deafness Sweating and dehydration Hyperventilation · Cutaneous flushing Hyperpyrexia (particularly children) Severe poisoning can cause convulsions, cerebral oedema, coma, renal failure, non-cardiogenic pulmonary oedema and cardiovascular instability. Arterial blood gas typically shows a respiratory alkalosis early in the course of the overdose due to hyper-stimulation of the respiratory centre. A raised anion gap metabolic acidosis can occur later in the overdose, especially in moderate to severe overdose, due to increased protons in the blood. Treatment involves stabilization of the ABCs as necessary, limiting absorption, enhancing elimination, correcting metabolic abnormalities, and providing supportive care. No specific antidote is available for salicylates. Gastric lavage and activated charcoal (50 g) are indicated if greater than 4.5 g has been ingested in the previous hour (or > 2 g in a child). Activated charcoal both reduces absorption and increases elimination of salicylate. Investigations should include: · Plasma salicylate level Arterial blood gas ECG Blood glucose level · Urea and electrolytes Clotting profile ECG abnormalities that can be present include: Widening of the QRS complex AV block · Ventricular arrhythmias Poisoning can be classified as mild, moderate or severe depending upon the plasma salicylate level: Mild poisoning = < 450 mg/L</li> Moderate poisoning = 450-700 mg/L Severe poisoning => 700 mg/L Severe cases usually require aggressive intravenous fluids to correct dehydration and 1.26% sodium bicarbonate administration, which increases elimination of the salicylate. The urine pH should be maintained at greater than 7.5 and ideally should be between 8.0-8.5. There is, however, no longer any role for forced alkaline diuresis. Life-threatening cases will require intensive care admission, intubation and ventilation and possibly haemodialysis.

CONTINUOUS CONTINUES TEATURES INCLUDE.

Salicylate poisoning is a relatively common cause of poisoning and effective early treatment can prevent organ damage and death.

# Common clinical features include:

- Nausea and vomiting
- Tinnitus
- Deafness
- Sweating and dehydration
- Hyperventilation
- Cutaneous flushing
- Hyperpyrexia (particularly children)

Severe poisoning can cause convulsions, cerebral oedema, coma, renal failure, non-cardiogenic pulmonary oedema and cardiovascular instability.

Arterial blood gas typically shows a respiratory alkalosis early in the course of the overdose due to hyperstimulation of the respiratory centre. A raised anion gap metabolic acidosis can occur later in the overdose, especially in moderate to severe overdose, due to increased protons in the blood.

## ECG abnormalities that can be present include:

- · Widening of the QRS complex
- AV block
- Ventricular arrhythmias

Erythromycin and the other macrolide antibiotics are bacteriostatic. They act by binding to the 50S subunit of the bacterial ribosome and inhibit translocation and therefore inhibit protein synthesis.

Macrolide antibiotics are actively concentrated within leukocytes, and are therefore transported into the site of infection.

Erythromycin is orally active and can also be administered intravenously. It has a biological half-life of 1.5 hours and is metabolised in the liver and excreted in the bile.

It has a similar antimicrobial spectrum to benzylpenicillin (i.e. narrow spectrum, mainly against **Grampositive organisms**) and can be used as an alternative in patients with penicillin allergy.

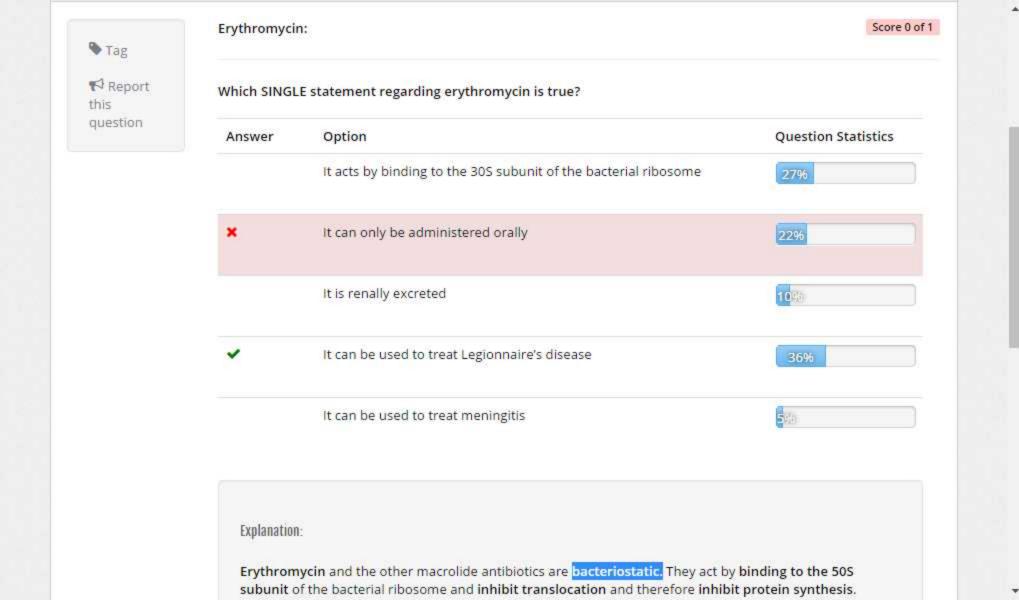
Erythromycin is ineffective in meningitis because it does not adequately penetrate the central nervous system. Unlike penicillin, it is effective against several atypical organisms and can be used to treatment infections with *Mycoplasma pneumoniae* and *Legionella pneumophila*.

### Common side effects of erythromycin include:

- Nausea and vomiting
- Abdominal pain
- Diarrhoea

### Rare side effects of erythromycin include:

- · Prolongation of the Q interval
- Arrhythmias (including torsades de pointes)
- Reversible deafness
- Cholestasis
- Steven-Johnson syndrome
- Toxic epidermal necrolysis





Report Report this question

### Meningitis:

Score 1 of 1

You review a 36-year-old lady whose daughter was recently admitted to a Paediatric Intensive Care Unit with meningococcal meningitis. She cared closely for her daughter in the period prior to her admission and is concerned about the possibility of her also contracting the disease. She is currently 22 weeks pregnant.

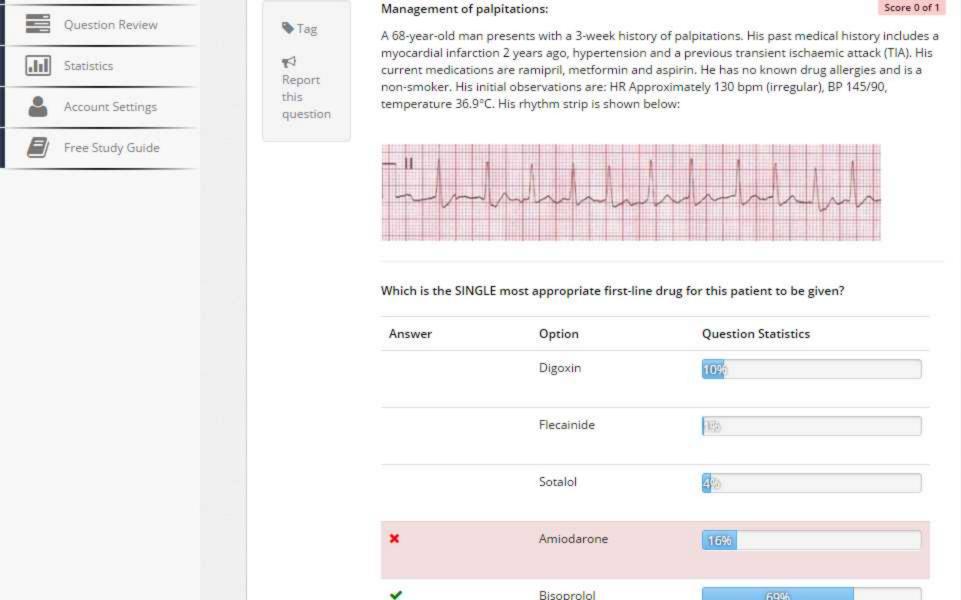
Which of the following antibiotics would be the MOST appropriate choice for chemoprophylaxis in her case? Select ONE answer only.

Answer	Option	Question Statistics
	Chloramphenicol 250 mg PO BD for 2 days	794
~	Ceftriaxone 250 mg IM	40%
	Rifampicin 600 mg PO BD for 2 days	28%
	Ciprofloxacin 500 mg PO	13%
	Penicillin V 500 mg QDS for 7 days	139

~	Ceftriaxone 250 mg IM	.40%
	Rifampicin 600 mg PO BD for 2 days	28%
	Ciprofloxacin 500 mg PO	139
	Penicillin V 500 mg QDS for 7 days	1390

For contacts of patients with *Neisseria meningitidis* meningitis the chemoprophylaxis agent of choice is rifampicin 600 mg PO BD for 2 days. A single oral dose of ciprofloxacin 500 mg may also be given.

Rifampicin and ciprofloxacin are both contraindicated in pregnancy, however, and cannot be given in this case. The agent of choice in this case is therefore a single 250 mg dose of intramuscular ceftriaxone.



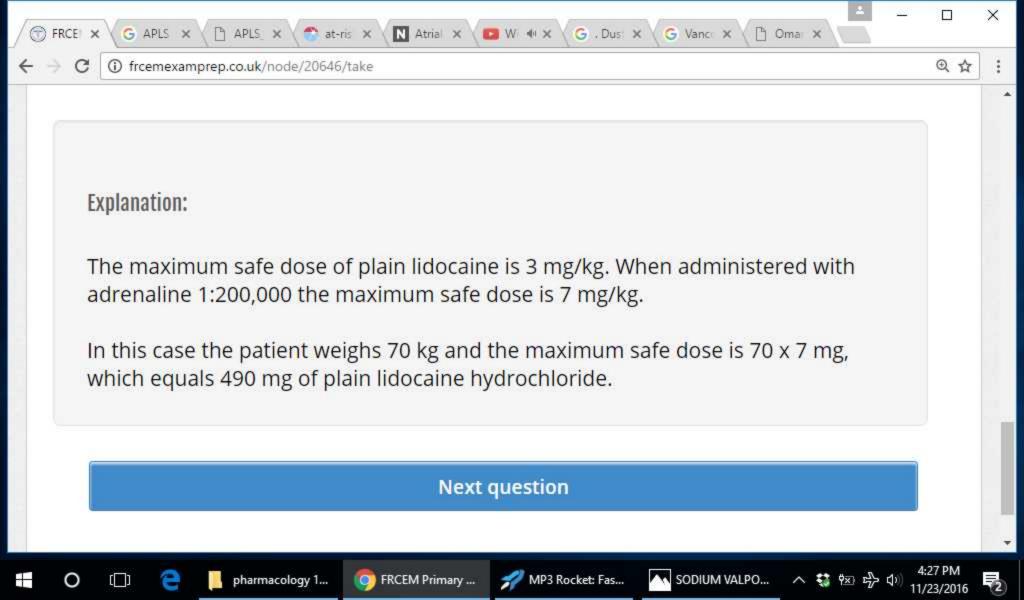
The diagnosis in this case is atrial fibrillation (AF), which seems to have started 3 weeks ago. This gentleman is over 65 and has a history of coronary artery disease, making him most suitable for a rate-control strategy for the management of his AF.

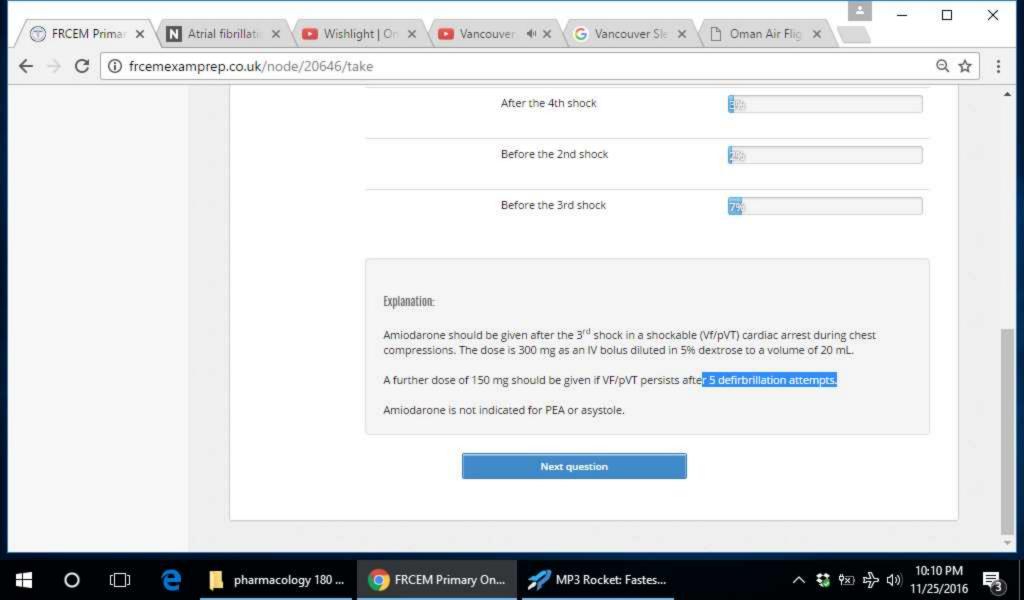
His past medical history makes him a high-risk patient and he should receive appropriate thromboprophylaxis and have warfarin or a suitable alternative initiated. For patients with a rate-control strategy the first line-drug should be a standard beta-blocker, such as bisoprolol, or a rate-limiting calcium channel blocker, such as diltiazem. A resting heart rate of less than 90 bpm should be targeted for established AF and less than 110 bpm for those with recent-onset AF.

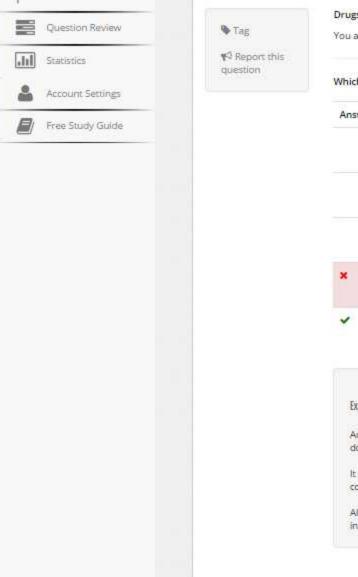
The use of digoxin is now reserved for patients requiring further rate-control therapy or for patients with co-existing heart failure.

Amiodarone, sotalol and flecainide are generally used when a rhythm control strategy has been adopted. Flecainide is generally best avoided in elderly patients with a history of coronary artery disease.

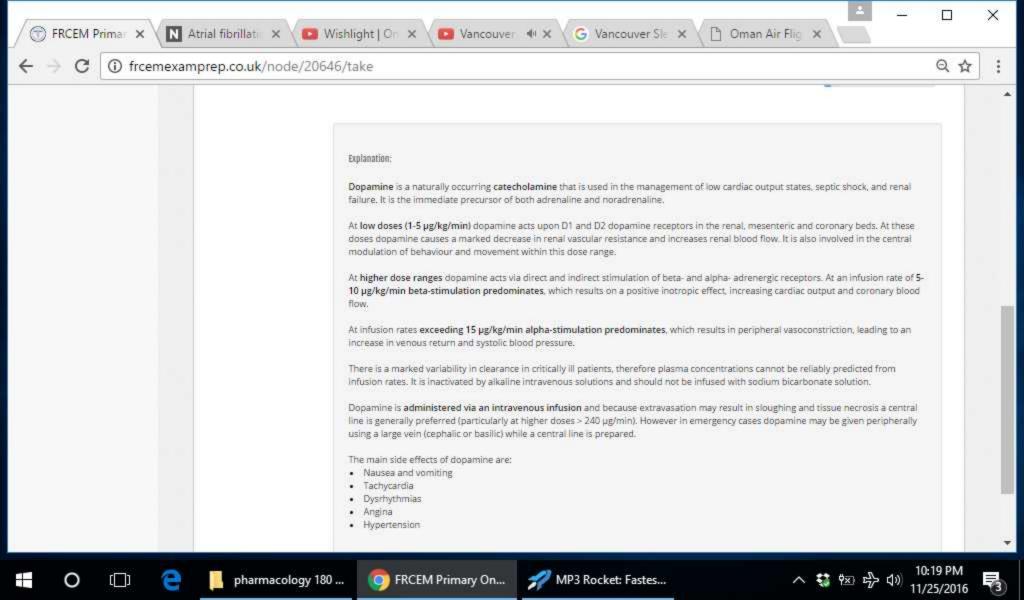
Please refer to the NICE guidelines on atrial fibrillation: www.nice.org.ukg/

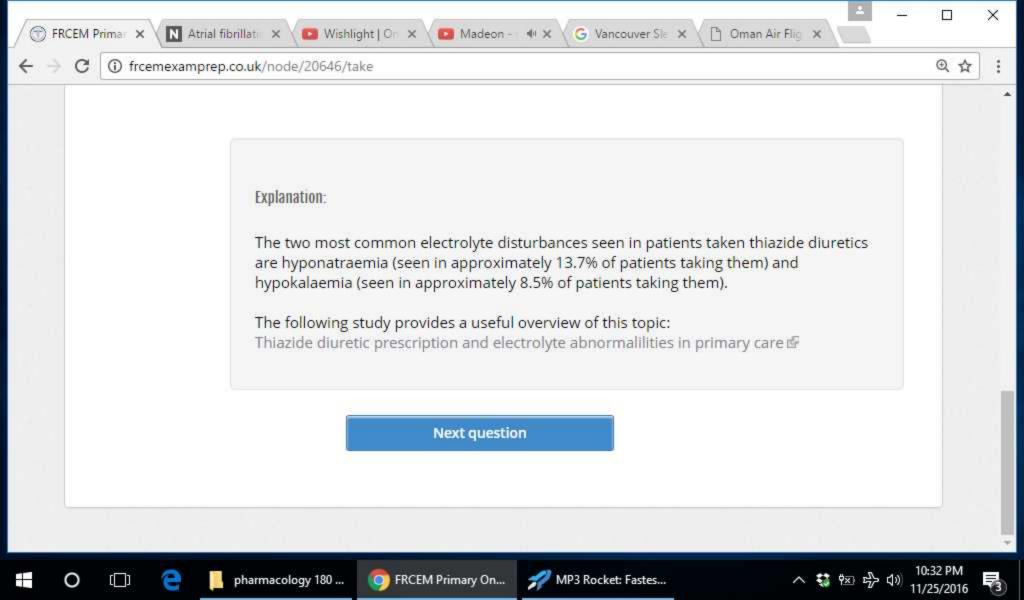






hich SINGLE	statement is true regarding the use of adrenaline in this arrest?	
Answer	Option	Question Statistics
	It cannot be given if intravenous access is unavailable	W)
	10 mL of 1:1000 solution is an appropriate dose	6 <sub>VA</sub>
	Chest compressions should be interrupted for administration	106
*	It should be administered every 2-3 minutes	5496
•	There is no evidence of long-term benefit from its use	38%
	e should be given as soon as circulatory access has been obtained in non-shocka ng (10 mL of 1:10,000 or 1 mL of 1:1000) via the IV or IO routes.	ble (PEA/asystole) cardiac arrests. The







Report this question

### Meningococcal meningitis:

Score 1 of 1

An 8-year-old girl presented to her GP with a headache, neck stiffness and photophobia. Her observations were as follows: HR 124, BP 86/43, RR 30, SaO<sub>2</sub> 95%, temperature 39.5°C. She has recently developed a petechial rash on her legs and arms. The GP administered a dose of antibiotics in the pre-hospital setting before the patient was transferred to the Emergency Department.

## Which ONE of the following would the GP have administered?

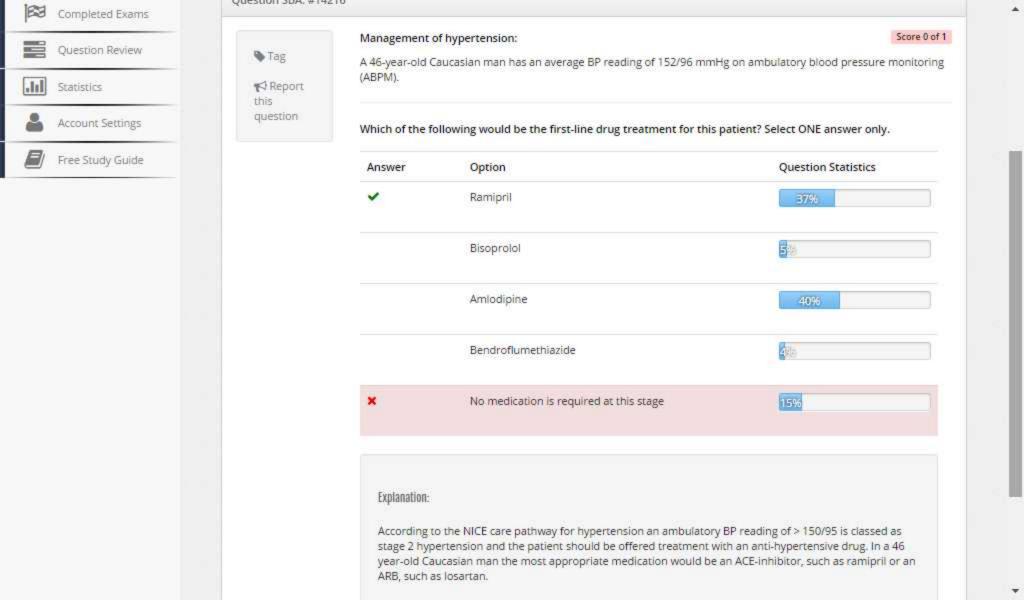
Answer	Option	Question Statistics
~	Give IM benzylpenicillin 600 mg	63%
	Give oral penicillin V 250 mg	
	Give oral penicillin V 500 mg	
	Give IM benzylpenicillin 300 mg	8%
	Give IM benzylpenicillin 1.2 g	24%

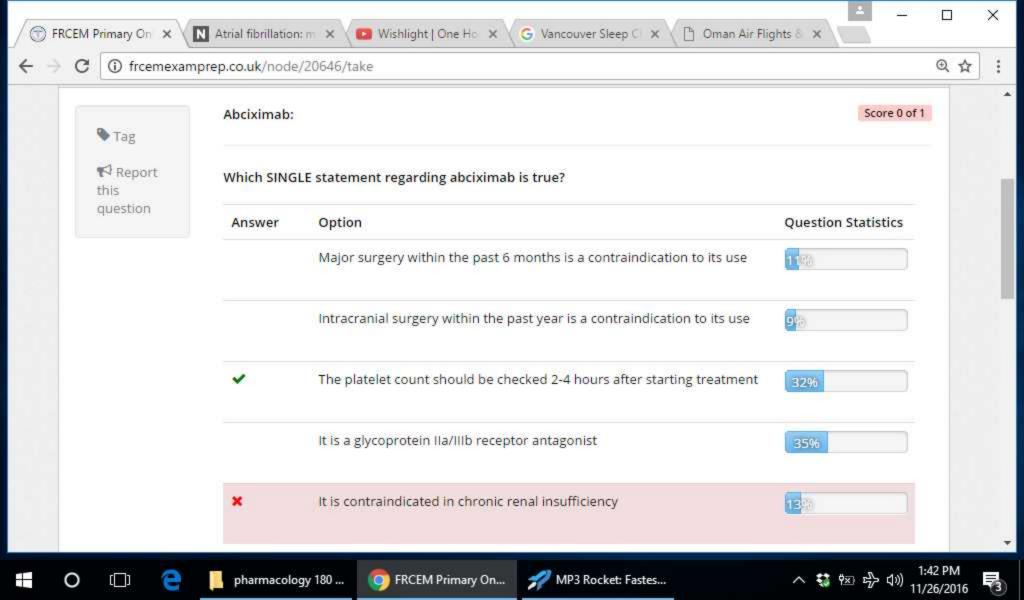
	Give IM benzylpenicillin 300 mg	8%
	Give IM benzylpenicillin 1.2 g	24%
Explanation:		

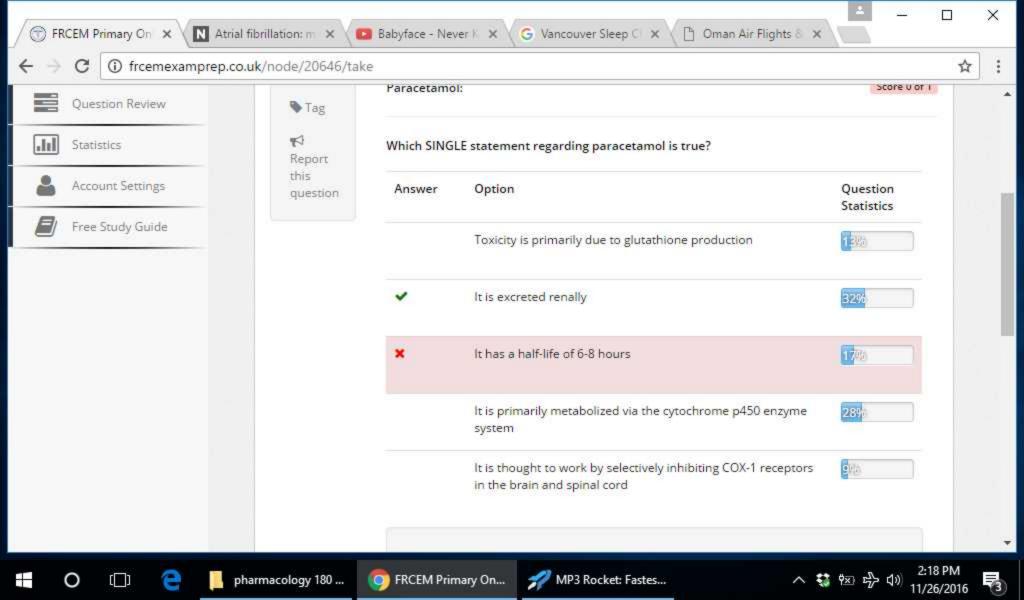
If bacterial meningitis and especially if meningococcal disease is suspected General Practitioners are advised to give a single injection of benzylpenicillin by intravenous or intramuscular injection before transferring the patient urgently to hospital.

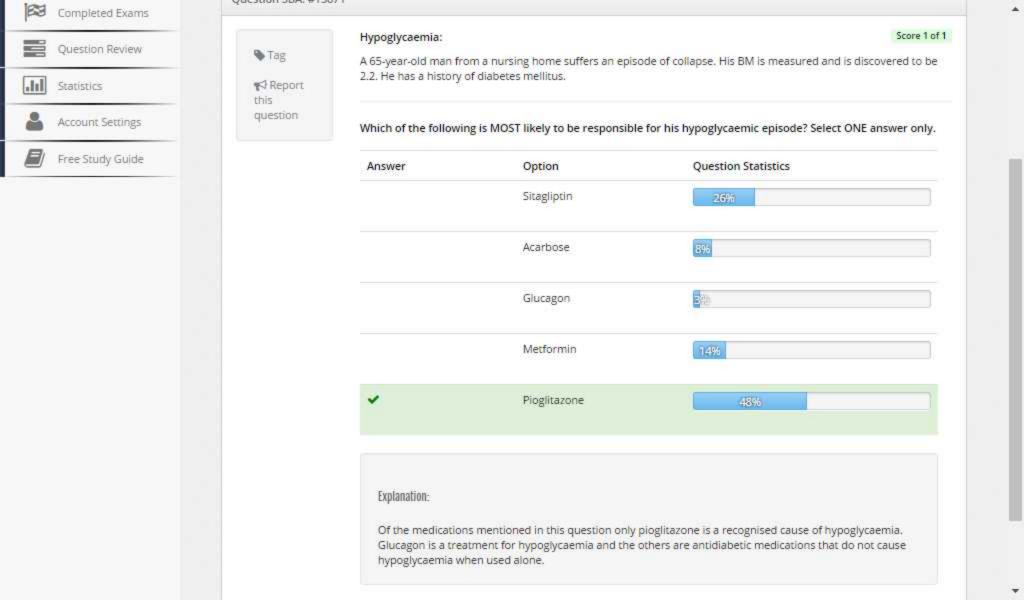
In children the following doses are recommended:

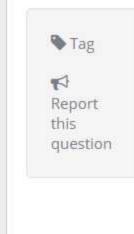
- . Infants under 1 year: 300 mg
- Children ages 1 to 9 years: 600 mg
- . Children aged 10 years and over 1.2 g











Whooping cough:

Angwar

A 6-year-old boy is diagnosed as having whooping cough. There are two members of the household that are considered to be within a 'priority group' for post-exposure chemoprophylaxis.

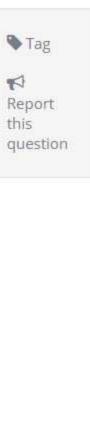
Score 1 of 1

Which of the following is the MOST appropriate antibiotic to be prescribed for this purpose? Select ONE answer only.

Ontion

Question Statistics

Answer	Option	Question Statistics
	Penicillin V	1296
	Co-amoxiclav	598
	Ciprofloxacin	9%
•	Erythromycin	58%
	Rifampicin	16%



Calcium channel blockers:

Which of the following statements regarding calcium channel blockers is FALSE? Select ONE answer only.

Score 0 of 1

Answer	Option	Question Statistics
~	They can be used in the treatment of heart failure	31%
	They can be used to treat migraine	1295
	They are associated with an increased risk of cardiac events	179
	They increase the risk of gastrointestinal bleeding	34%
×	They commonly cause ankle oedema	600

Calcium channel blockers prevent the movement of calcium into cells via the L-type calcium channel. This results in the relaxation of vascular smooth muscle in vessel walls and a resultant reduction in peripheral vascular resistance.

They have a variety of uses including:

- Hypertension
- Angina
- Atrial fibrillation
- Migraine

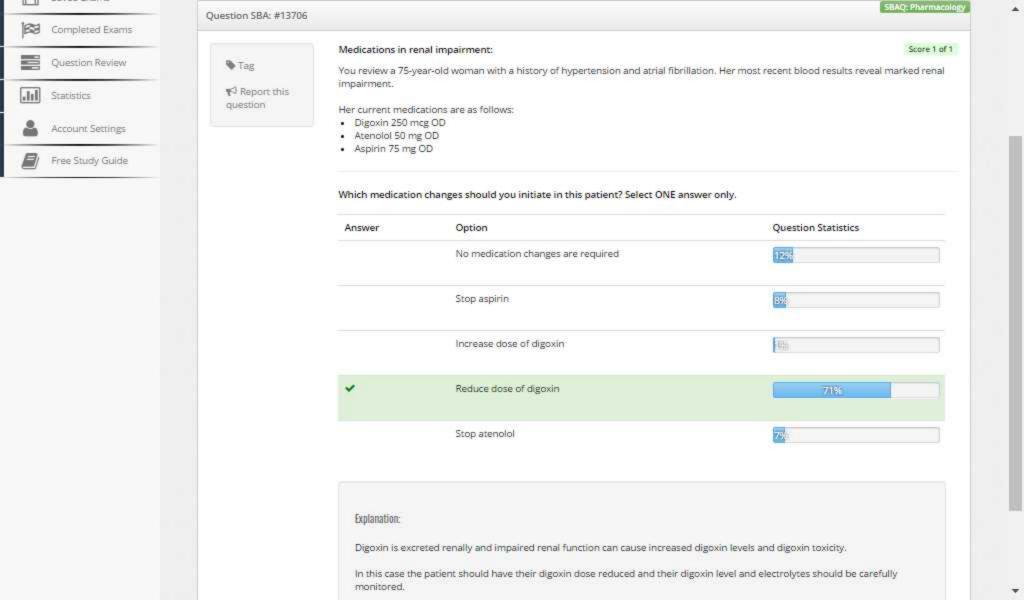
Short acting dihydropyridine calcium channel blockers have been shown to increase cardiac mortality and cardiac events in patients with coronary heart disease. This is not true of long acting calcium channel blockers.

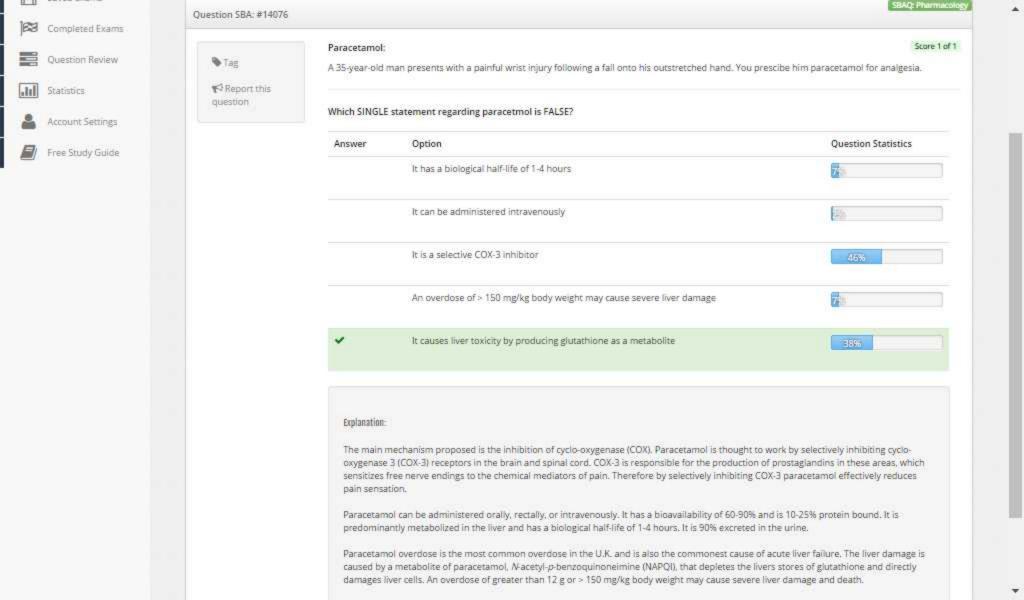
Calcium channel blockers have been found to be moderately useful in the prevention of migraines. The best evidence is for this is with verapamil. This may be due to prevention of the arteriolar constriction that is associated with migraine. They are commonly used for this elsewhere in the world but are not currently licensed for this use in the UK.

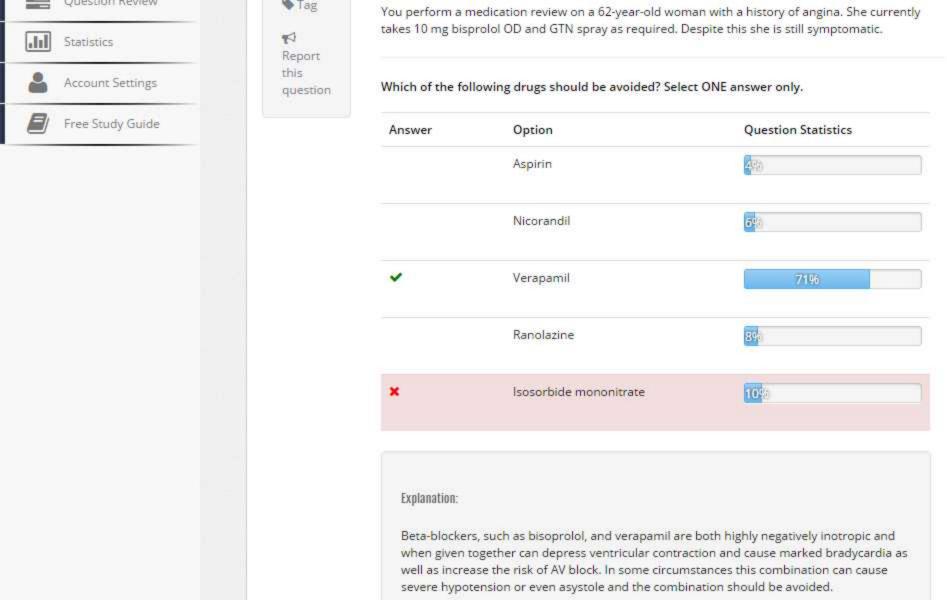
Calcium channel blockers have a number of common side effects including:

- Constipation
- Flushing
- Headaches
- Ankle oedema
- Palpitations

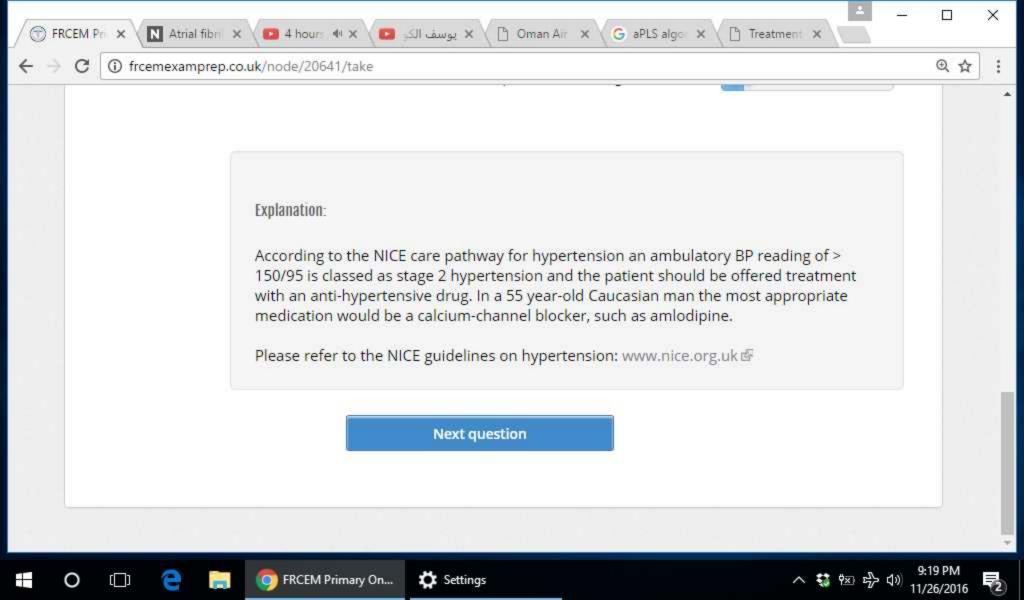
Calcium channel blockers are also associated with an increased risk of gastrointestinal bleeding.

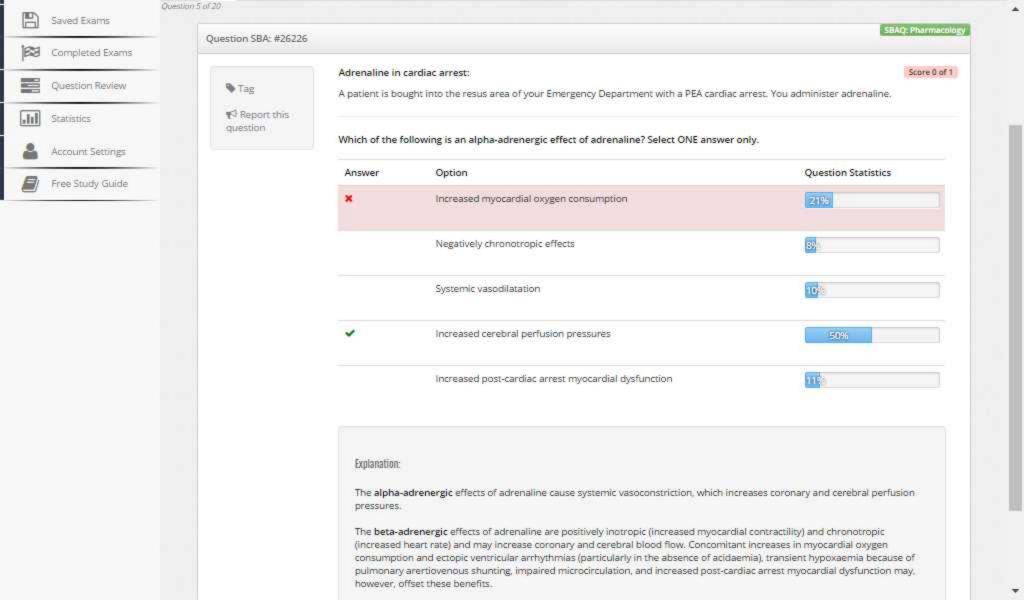


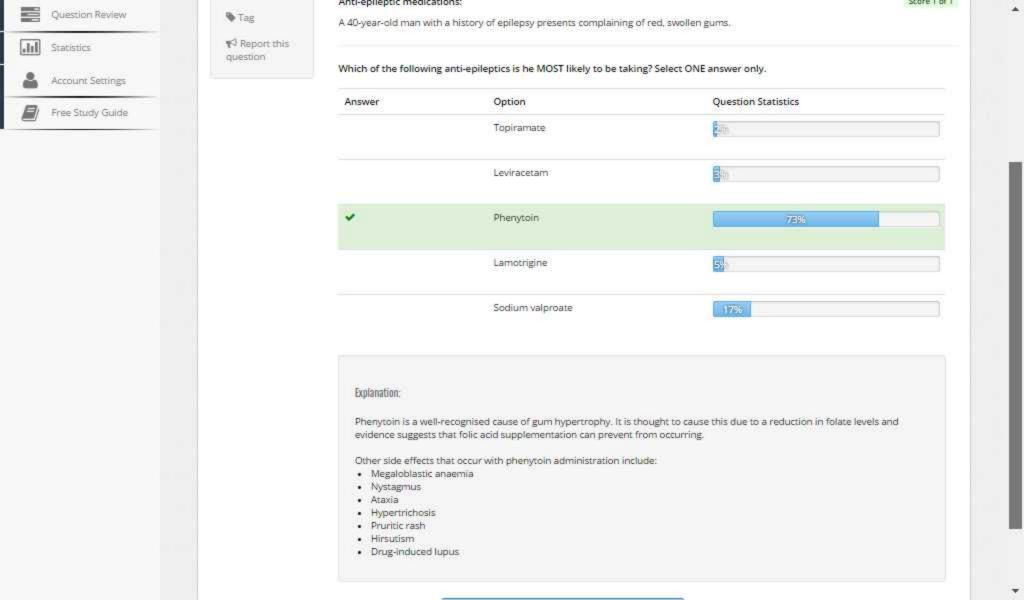




¥









## Explanation:

Anti-D is an IgG class antibody directed against the Rhesus D (RhD) antigen.

Anti-D is only given to RhD negative women. RhD negative women do not carry the RhD antigen on their RBC. If a fetus does carry the RhD antigen (i.e. is RhD positive) and the mother is exposed to fetal blood, she may form antibodies to RhD that pass through the placenta to attack fetal red cells (causing haemolytic disease of the newborn) in this or subsequent pregnancies. Anti-D is given to bind fetal red cells in the maternal circulation to neutralise them before an immune response is triggered.

RhD should be given in the event of a sensitising event. Potentially sensitising events include:

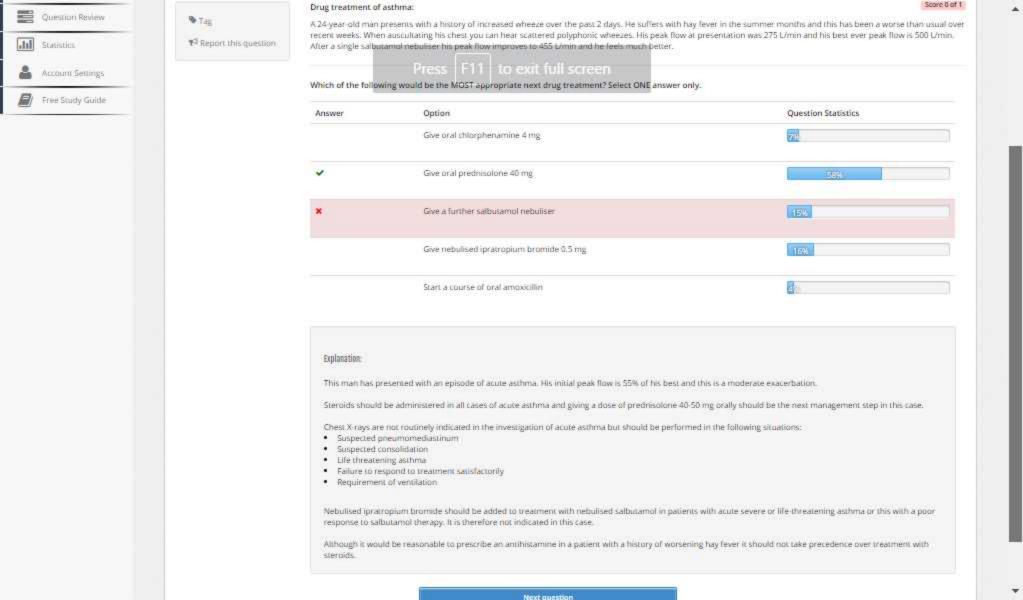
- · Birth
- Antepartum haemorrhage
- Miscarriage
- · Ectopic pregnancy
- · Intrauterine death
- Amniocenetsis
- Chorionic villus sampling
- · Abdominal trauma

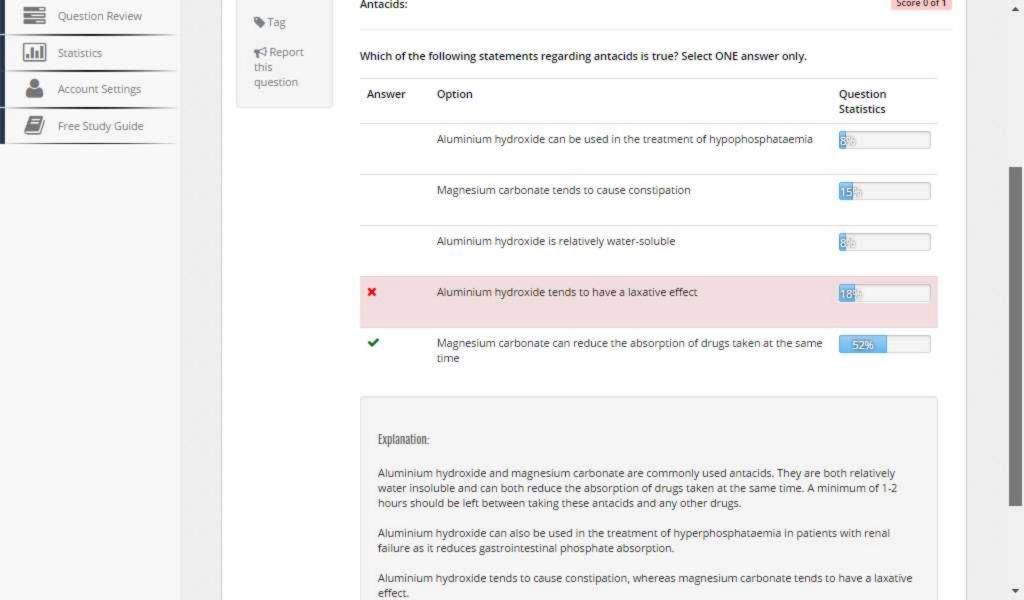
It the event of a sensitising event occurring, the sooner anti-D is given the better, but it is maximally effective within 72 hours and the BNF states it is still likely to have some benefit if administered outside of this deadline.

Routine antenatal prophylaxis is recommended for RhD negative women at 28 and 34 weeks. This is irrespective of whether they have already received Anti-D earlier in the same pregnancy for a sensitising event.

Before 12 weeks gestation, confirmed by scan, in uncomplicated miscarriage (where the uterus is not instrumented), or mild painless vaginal bleeding, prophylactic anti-D is not necessary because the risk of feto-maternal haemorrhage (FMH) is negligible. However 250 IU of prophylactic anti-D immunoglobulin should be given in cases of therapeutic termination of pregnancy, whether by surgical or medical methods, to confirmed RhD negative women who are not known to be already sensitised to RhD.

	Doxcycline 100 mg PO OD for 7 days	894
	Amoxicillin 500 mg PO TDS for 7 days	735Va
	No treatment is required in this case	
Explanation:		
This patient had infection.  The clinical fea  Flu-like illne  Fever  Myalgia  Headache  Diarrhoea  Cough (init)	s symptoms and signs consistent with an atypical pneumonia.  Itures of Mycoplasma pneumoniae infection include: ess preceding respiratory symptoms  ially dry but often becomes productive)	nost likely secondary to <i>Mycoplasma pneumoniae</i>
infection.  The clinical fea Flu-like illow Fever Myalgia Headache Diarrhoea Cough (init Focal chest	tures of <i>Mycoplasma pneumoniae</i> infection include: ess preceding respiratory symptoms	





C1 esterase inf	fusion	36%	
IM a <mark>d</mark> renaline		15%	
Explanation:			
Hereditary angioedema is caused by a complement system. It is usually inheri			the
Symptoms usually begin in childhood a	nd occur sporadically throug		

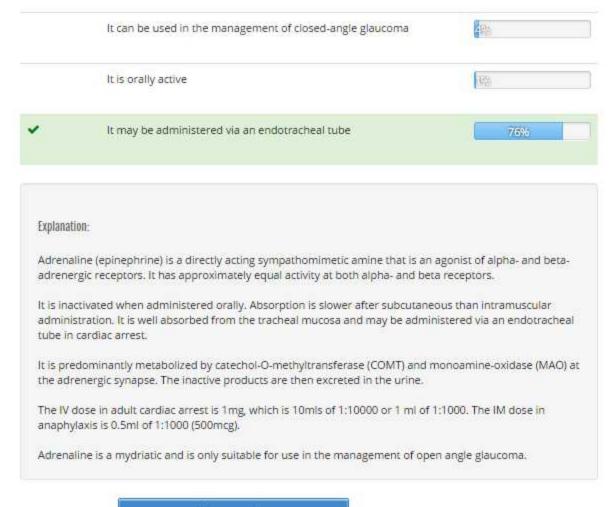
Symptoms usually begin in childhood and occur sporadically throughout adult life. Attacks can be precipitated by minor surgical and dental procedures and stress. The main clinical features of hereditary angioedema are oedema of the skin and mucous membranes. The most commonly affected areas are the face, tongue and extremities. There is often a prodrome of tingling and it is sometimes preceded by a non-pruritic rash.

Angioedema and anaphylaxis due to C1 esterase inhibitor deficiency is resistant to adrenaline, steroids and antihistamines and needs treatment with C1 esterase inhibitor concentrate or fresh frozen plasma, which contains C1 esterase inhibitor.

Short-term prophylaxis for situations that may precipitate an attack can be achieved with C1 esterase inhibitor or fresh frozen plasma infusions prior to the event.

Long-term prophylaxis can be achieved with androgenic steroids such as stanozolol or antifibrinolytic drugs such as tranexamic acid.

Next question



Next question

They are indicated in the long-term management of severe anxiety



## **Explanation**:

Benzodiazepines enhance the effects of GABA resulting in sedative, hypnotic, anxiolytic, anticonvulsant and muscle relaxant properties.

Benzodiazepines are indicated in the short-term management of severe anxiety, long-term use should be avoided due to potential problems with tolerance, physical dependence and withdrawal syndrome.

Diazepam is a long-acting benzodiazepine with a half-life of 20-100 hours. Examples of short-acting benzodiazepines with a half-life of less than 12 hours include midazolam, oxazepam and alprazolam (Xanax).

Oral lorazepam 1-2 mg or IM lorazepam 2-4 mg are recommended for the emergency tranquilization of violent or disturbed patients.

The dose of lorazepam in paediatric status epilepticus is 0.1 mg/kg.

Score 1 of 1

Question Statistics



51%











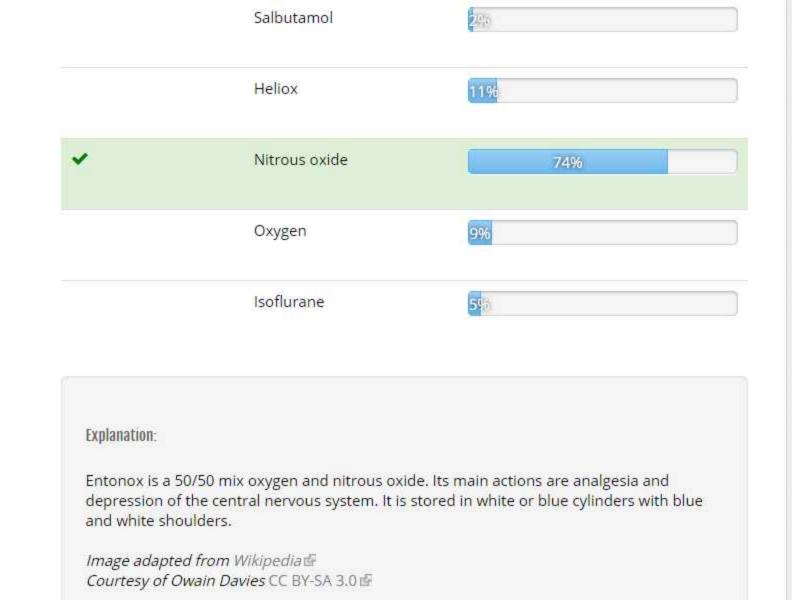


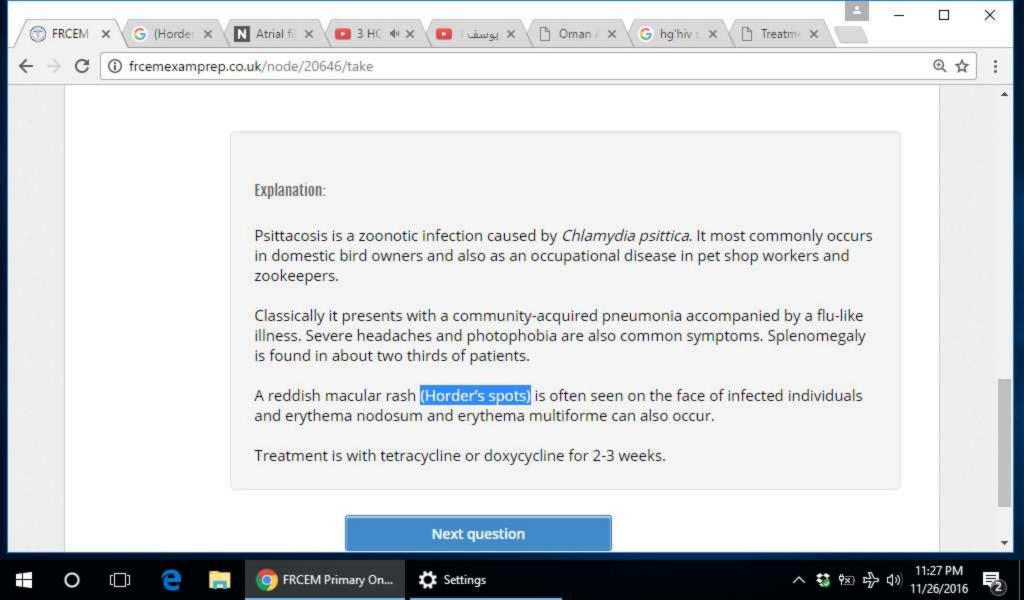
Report this question

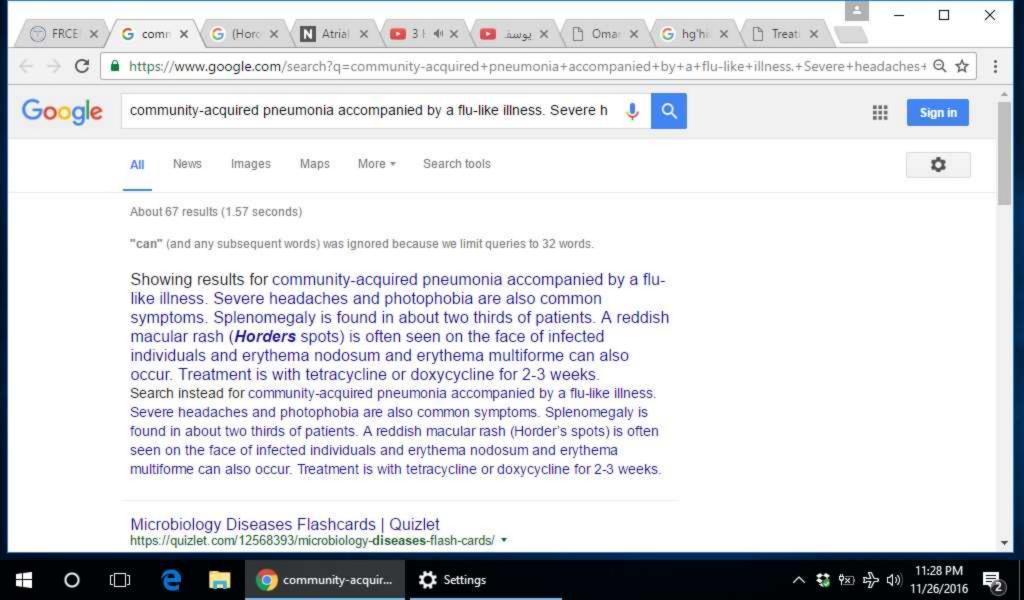
## Analgesia and anaesthesia in the ED:

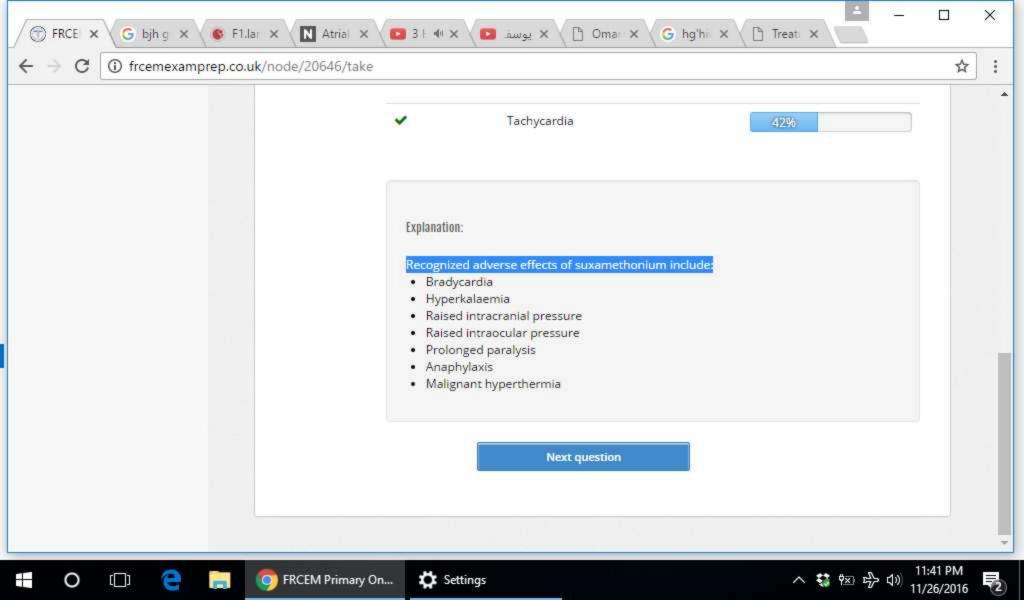
Please refer to the following cylinder that you have found in the A&E 'resus' area:

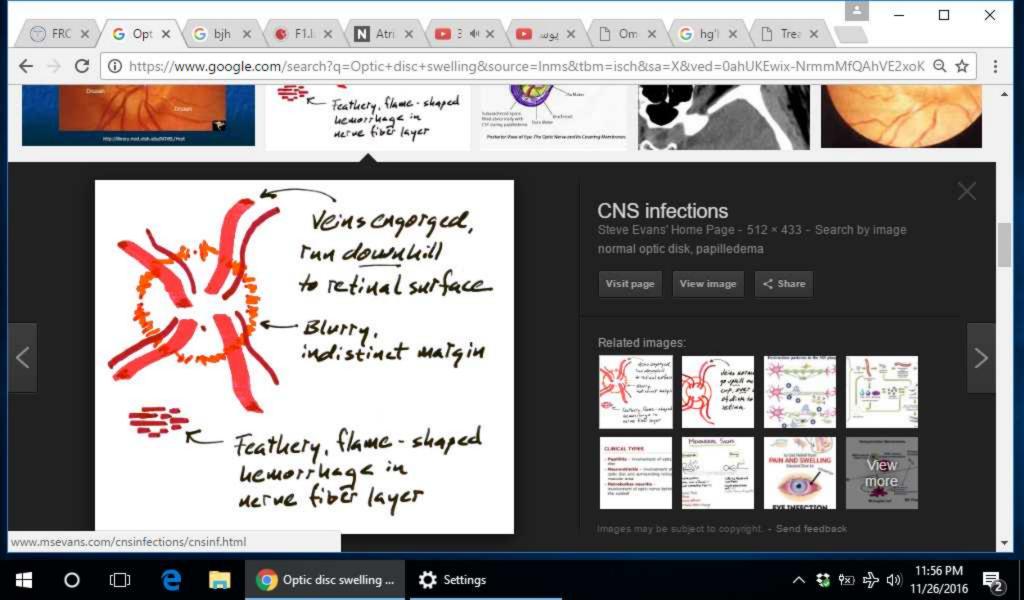


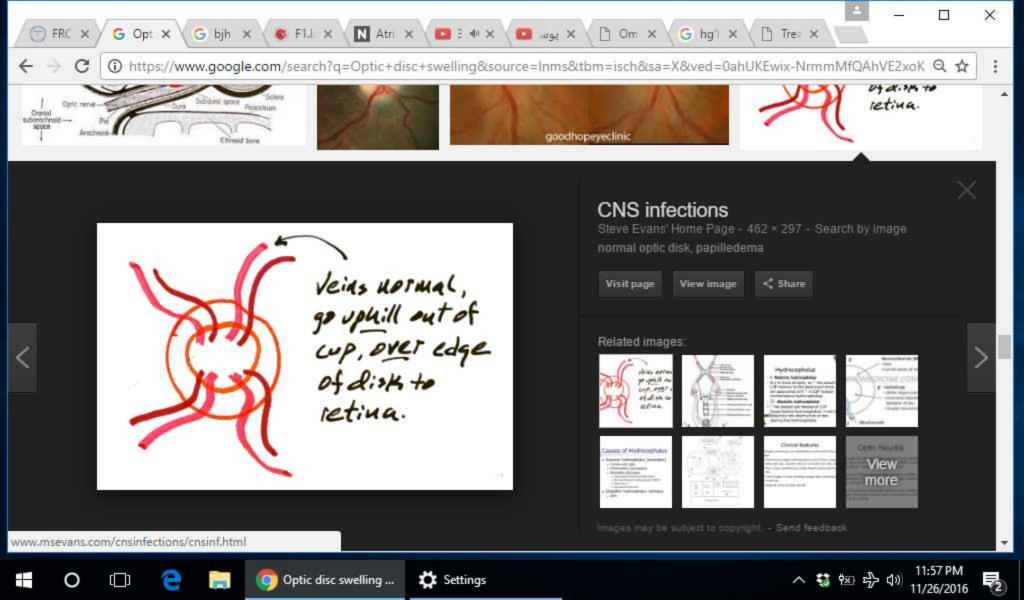


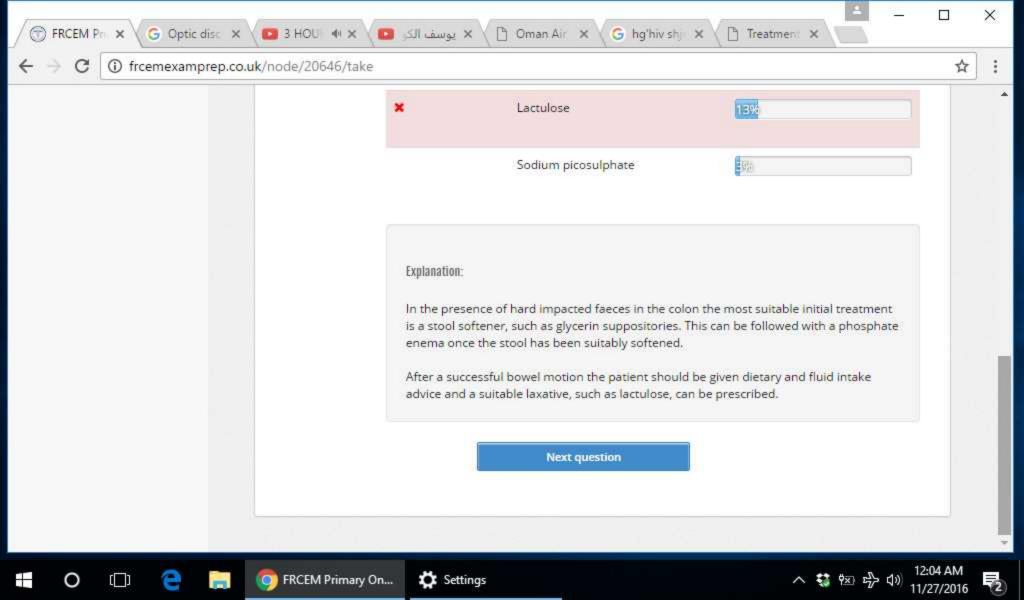


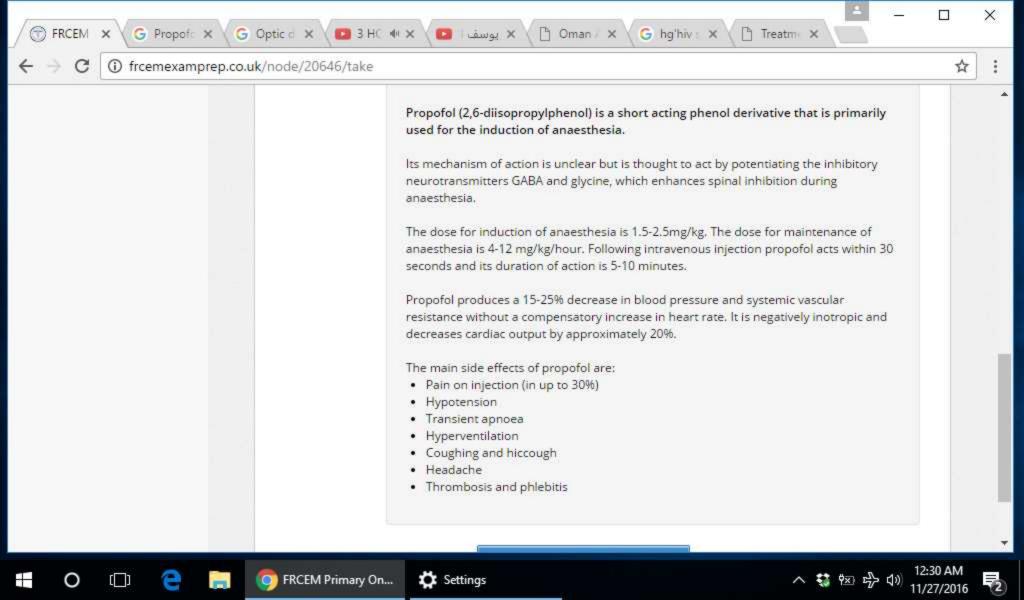


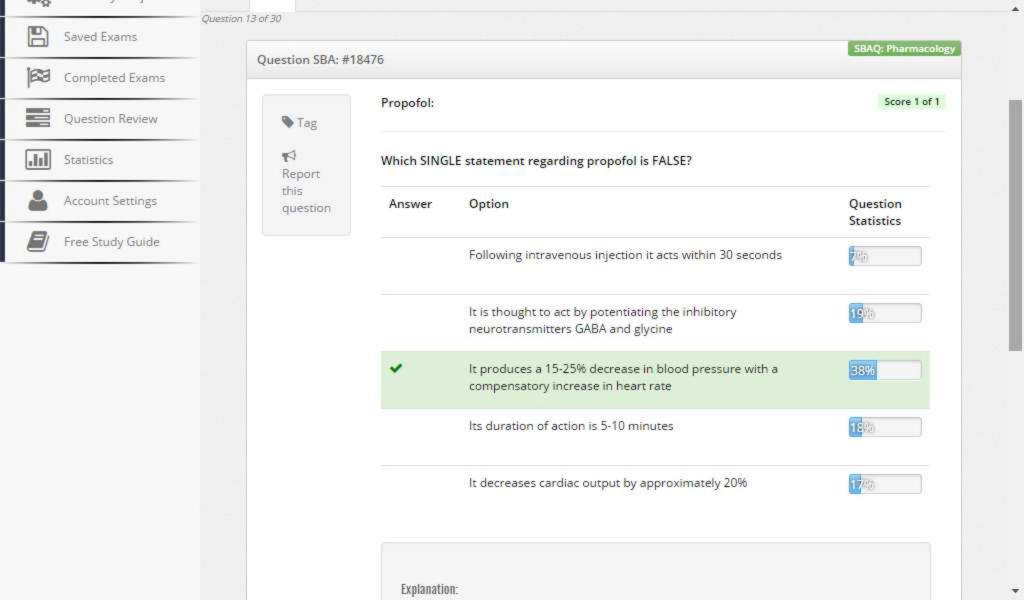


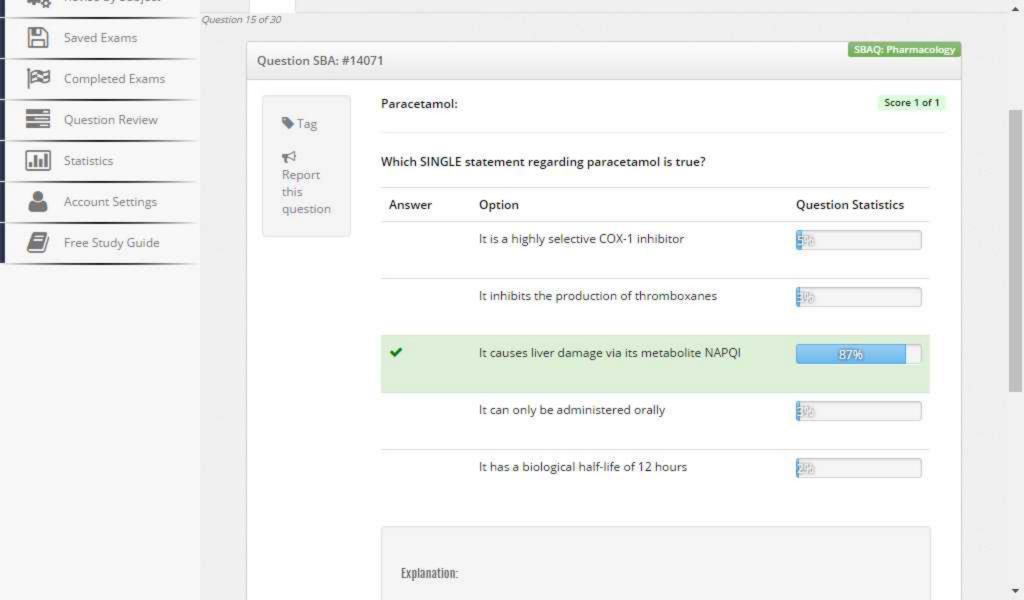


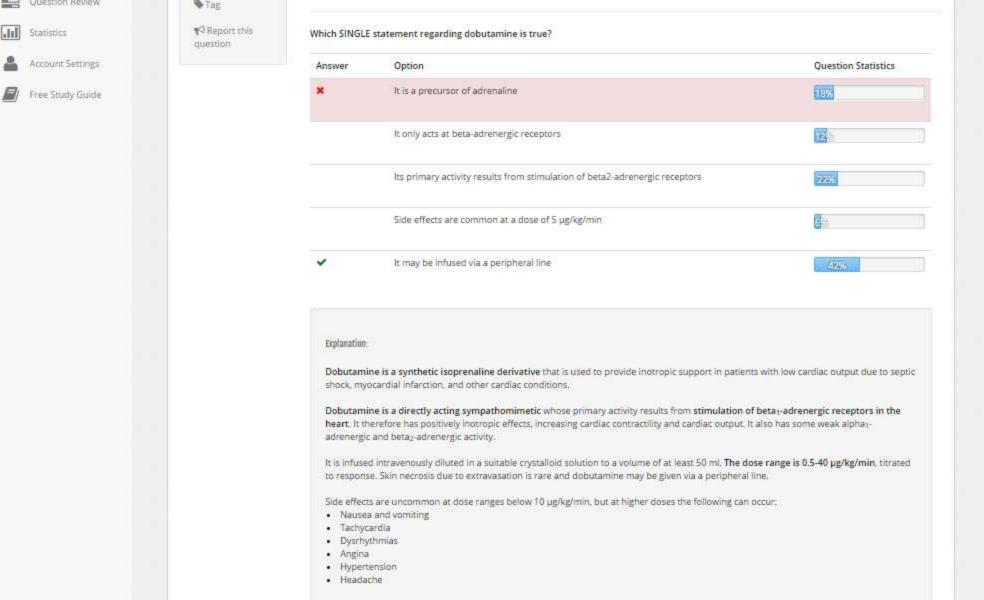




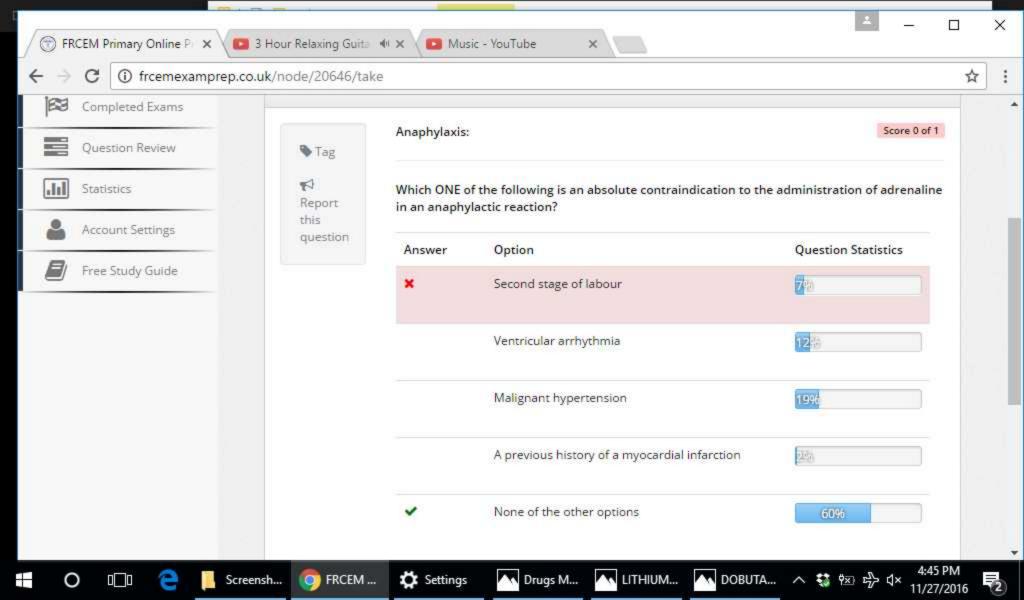


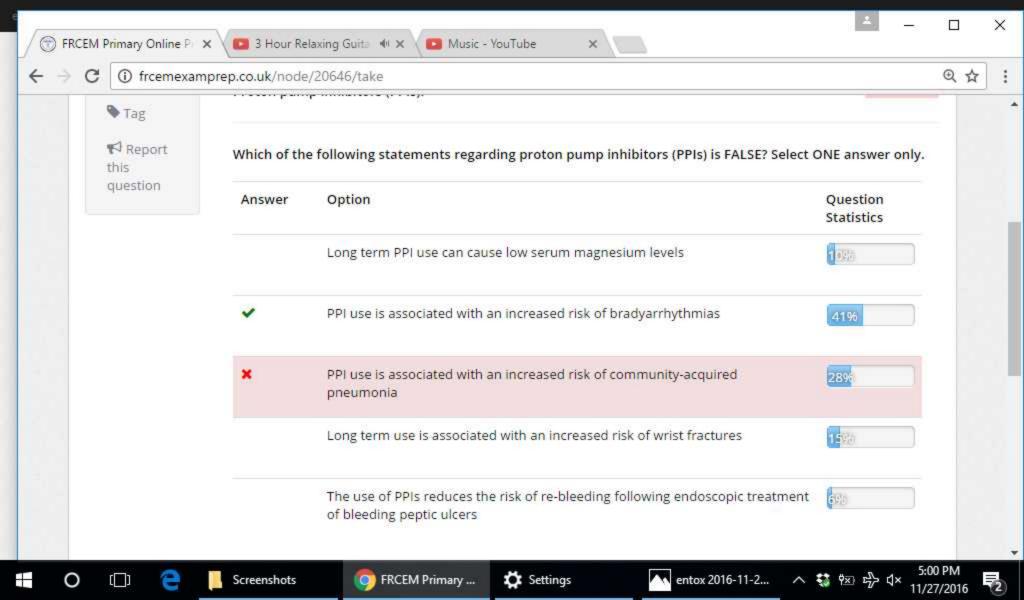


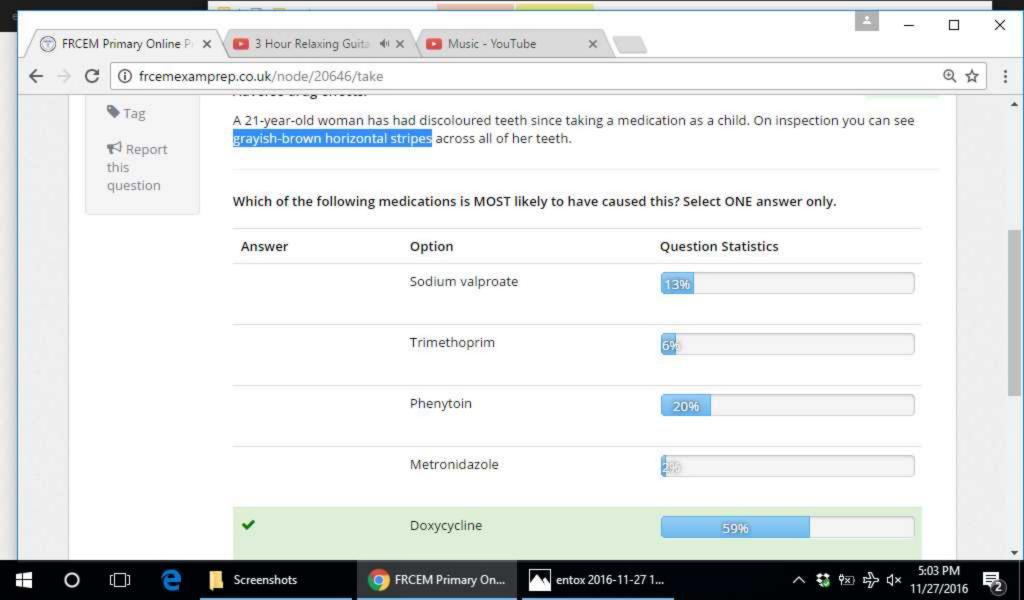


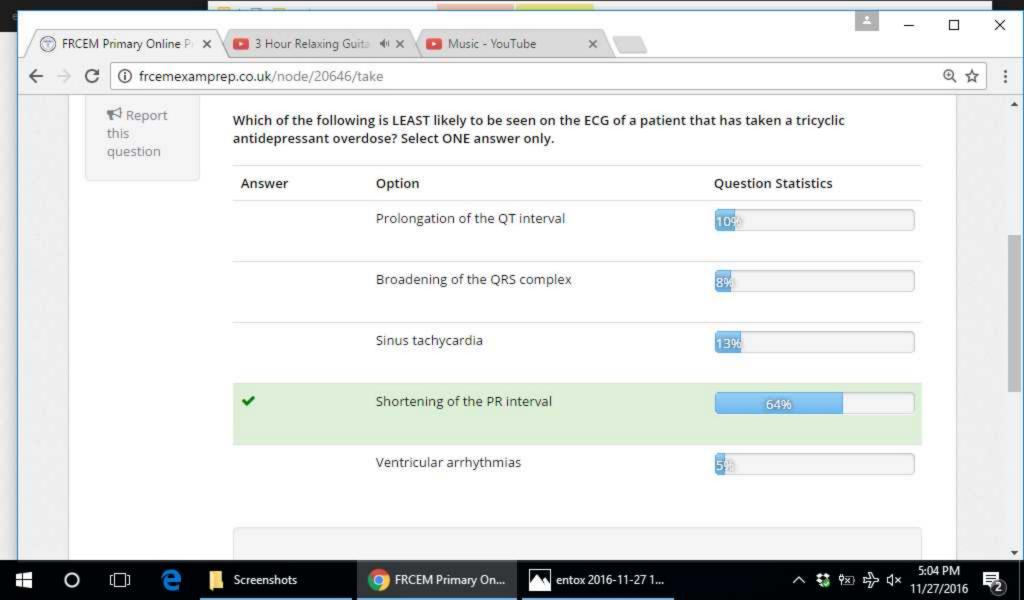


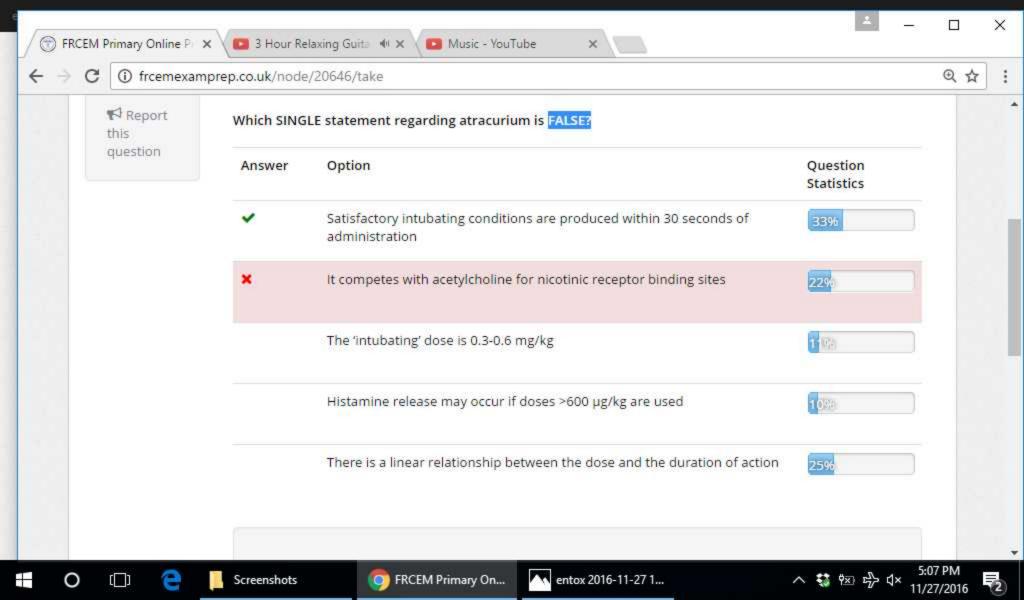
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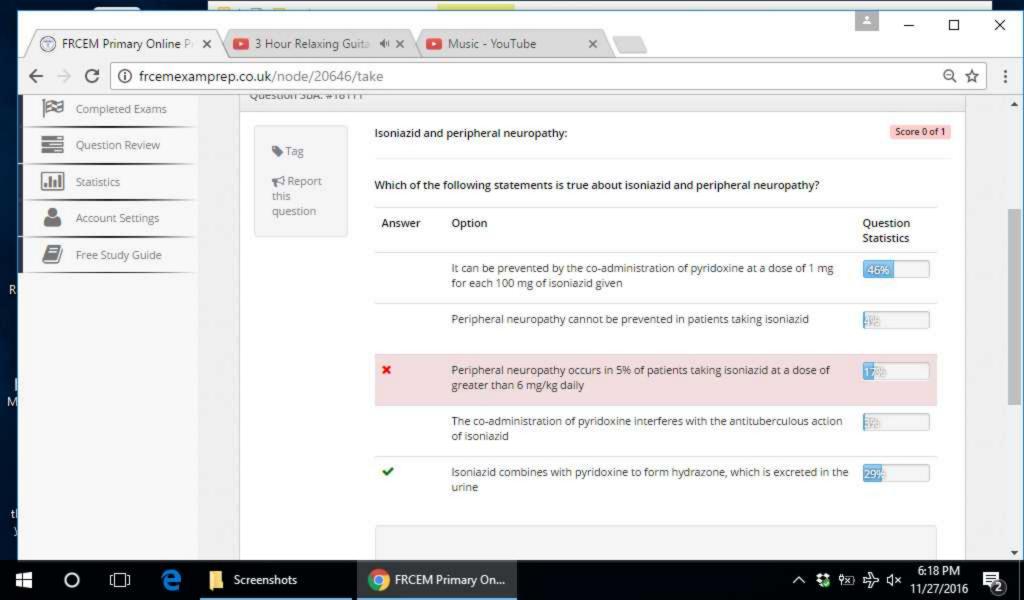


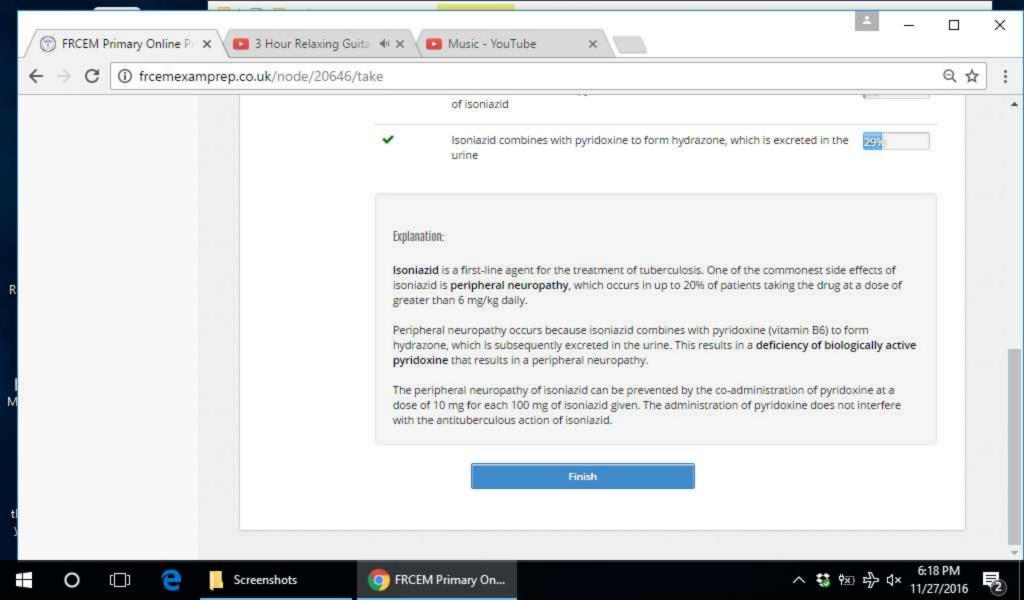


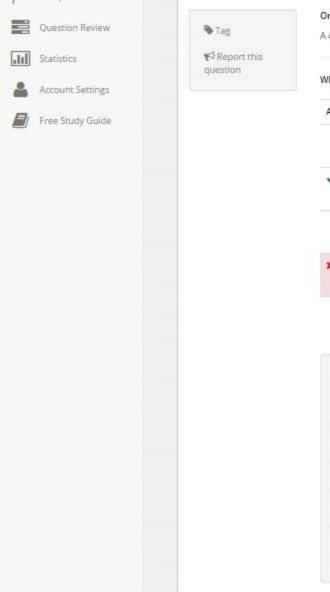












ral Rehydration Therapy (ORT):  4-year-old boy presents with viral gastroenteritis. You recommend treatment with oral rehydration therapy (ORT) e.g. dioralyte.					
Which of the following statements regarding the use of ORT in the management of gastroenteritis is FALSE? Select ONE answer only.					
Answer	Option	Question Statistics			
	50 ml/kg given over 4 hours is recommended for the treatment of mild dehydration	<b>B</b>			
~	ORT is sugar-free	42%			
	ORT is hypo-osmolar	30%			
×	To prevent dehydration a child with diarrhoea should drink 200 ml of ORT after each loose stool				
	ORT contains salts	<b>G</b> ia			
Explanation					
	luid replacement strategy used to prevent or treat dehydration, it is less invasive that other strategies for i tessful at lowering the mortality rate of diarrhoea in developing countries.	fluid replacement and has			
	ains glucose (e.g. 90 mmol/L in dioralyte). The addition of glucose improves sodium and water absorption aemia. It also contains essential mineral salts.	in the bowel and prevents			
Current 1	INCE guidance recommends that 50 ml/kg is given over 4 hours for the treatment of mild dehydration.				
	ydrated, a child should continue with their usual daily fluid intake plus 200ml ORT after each loose stool. In normal feed volume and in an adult give 200-400 mL after each loose stool.	n an infant give ORT at 1-			
For furth	er information refer to the NICE guidance on diarrhoea and vomiting caused by gastroenteritis in under 5s	: www.nice.org.uk dF			

The patient has had a previous history of anaphylaxis following their first BCG vaccination





The patient is asplenic



## Explanation:

All vaccines are recommended, whether live or inactivated, in patients with asplenia.

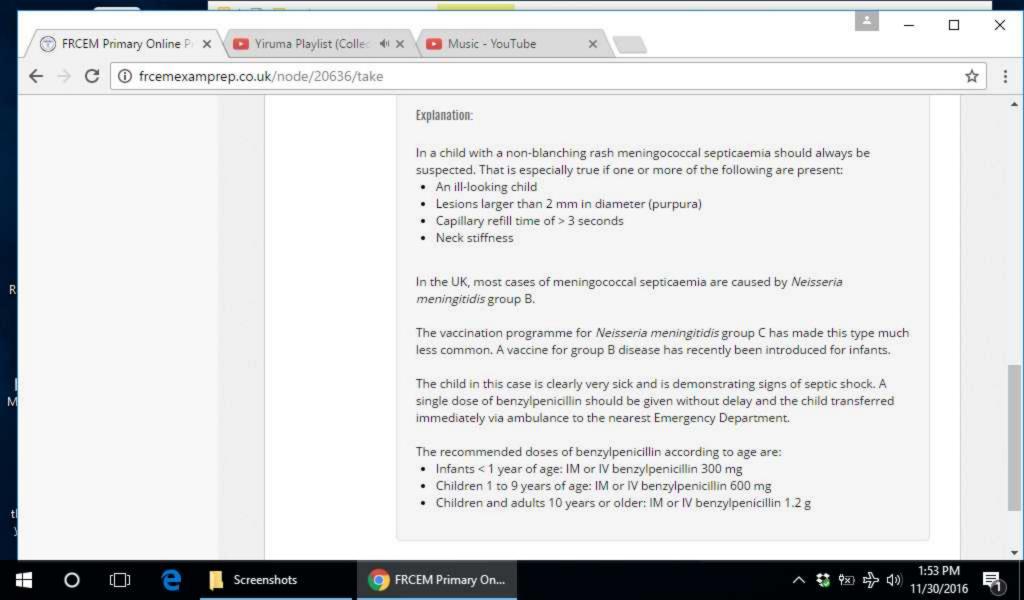
A history of anaphylaxis following any vaccination would be a contraindication to having that vaccine again.

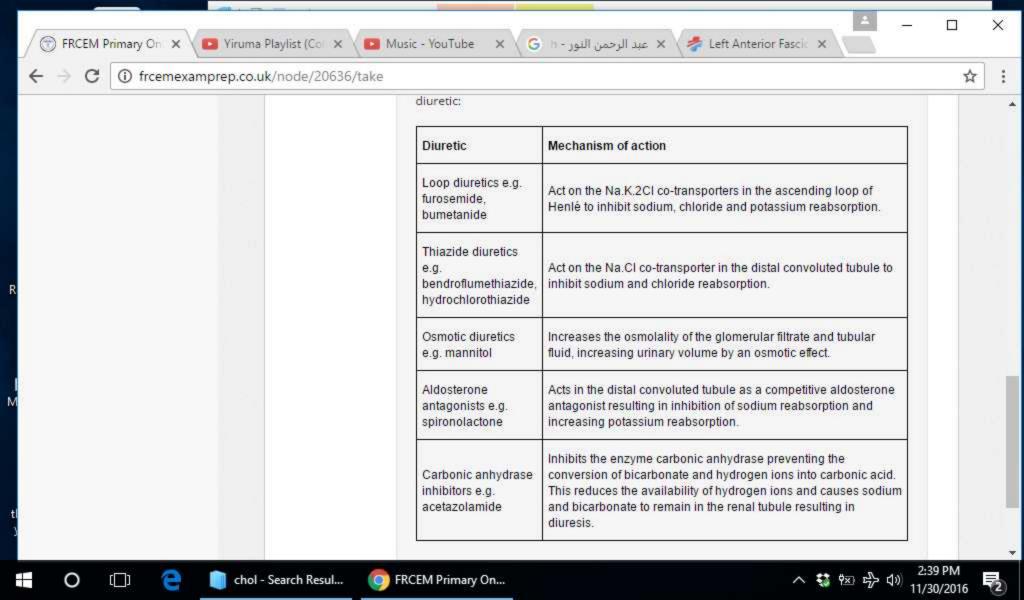
BCG is a live vaccine. Live vaccinations are generally contraindicated in the following situations:

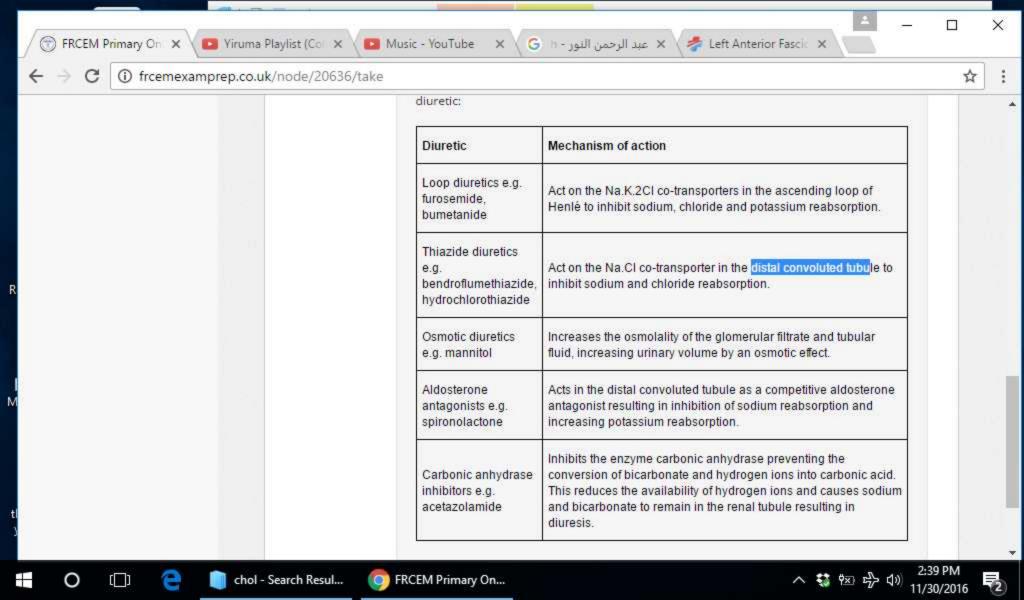
- Pregnancy
- · HIV, whether asymptomatic or symptomatic
- If less than 3 weeks after another live vaccine (although 2 live vaccinations can be given together at different sites of the body)
- . Other illnesses causing severe compromise of the immune system
- · Haematological malignancies

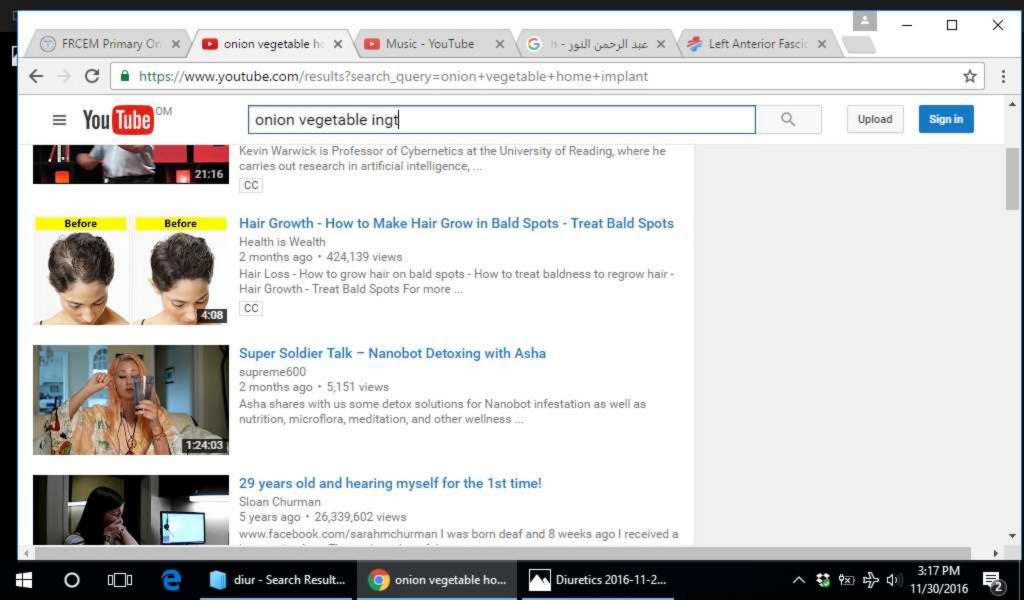
The CDC has some excellent guidelines that can be viewed here: www.cdc.gov ₽

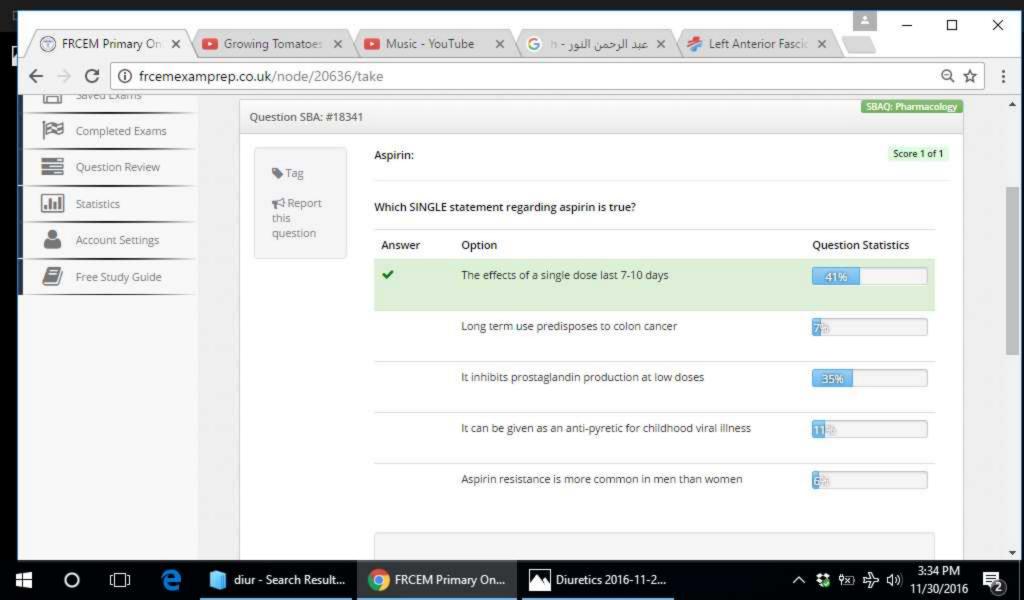
**Next question** 

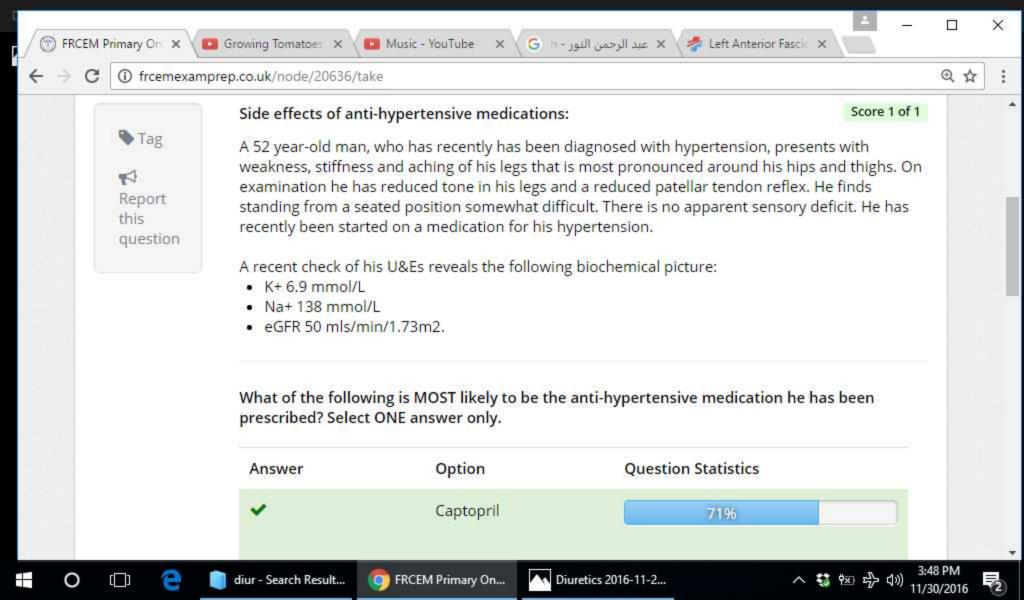












This patient has presented with symptoms and signs consistent with myopathy. Myopathy is characterised by:

- Muscle weakness
- Muscle atrophy
- Tone and reflexes can be reduced

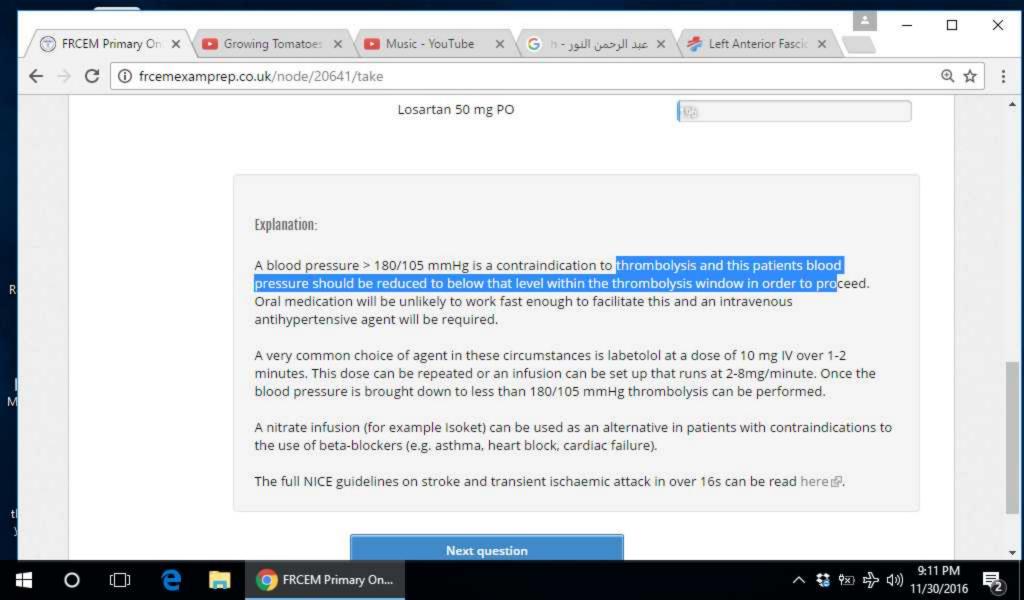
Hyperkalaemia is a known biochemical cause for myopathy. Other metabolic causes include:

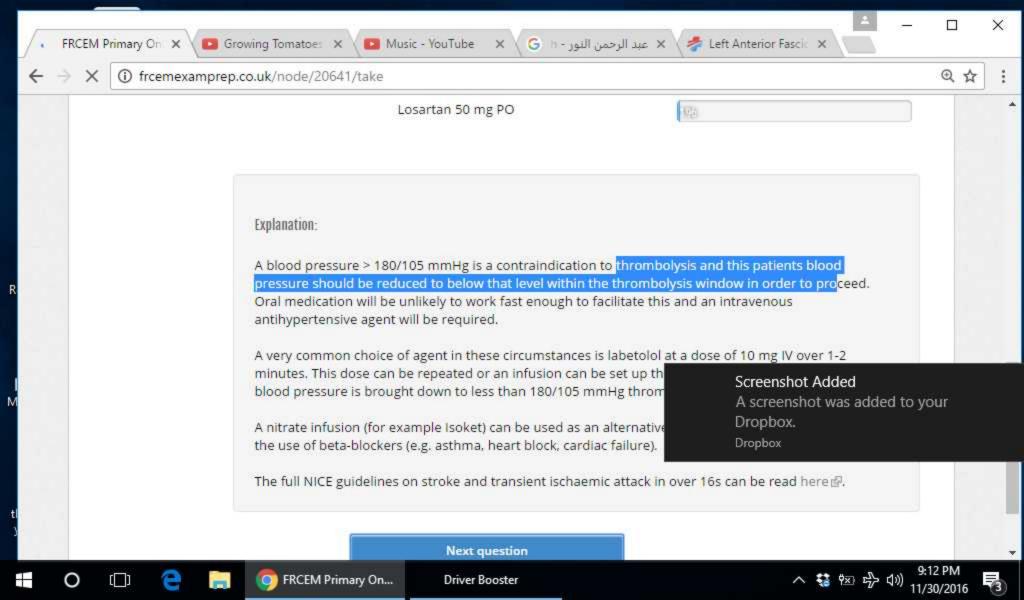
- Hypokalaemia
- Hypercalcaemia
- Hypomagnesaemia
- Hyperthyroidism
- Hypothyroidism
- Diabetes mellitus
- Cushing's disease
- Conn's syndrome

ACE inhibitors, such as captopril, are a well-recognised cause of hyperkalaemia and are likely to be the culprit in this case.

Other commonly encountered side effects of ACE inhibitors include:

- · Renal impairment
- · Persistent dry cough
- Angioedema (onset can be delayed)
- Rashes
- Upper respiratory tract symptoms including sore throat
- Gastrointestinal upset





Flecainide is a class ic antiarrhythmic agent that acts by blocking the Nav1.5 sodium channel in the heart, thereby prolonging the cardiac action potential and slowing conduction of the cardiac impulse within the heart. It has a profound effect on conduction in accessory pathways, especially on retrograde conduction, and markedly suppresses ventricular ectopic foci.

Flecainide can be used in the treatment of many different arrhythmias including:

- · Pre-excitation syndromes (e.g. Wolff-Parkinson-White)
- Acute atrial arrhythmias
- · Ventricular arrhythmias

It has also been shown to be effective in the treatment of chronic neuropathic pain, www.ncbl.nlm.nih.gov.

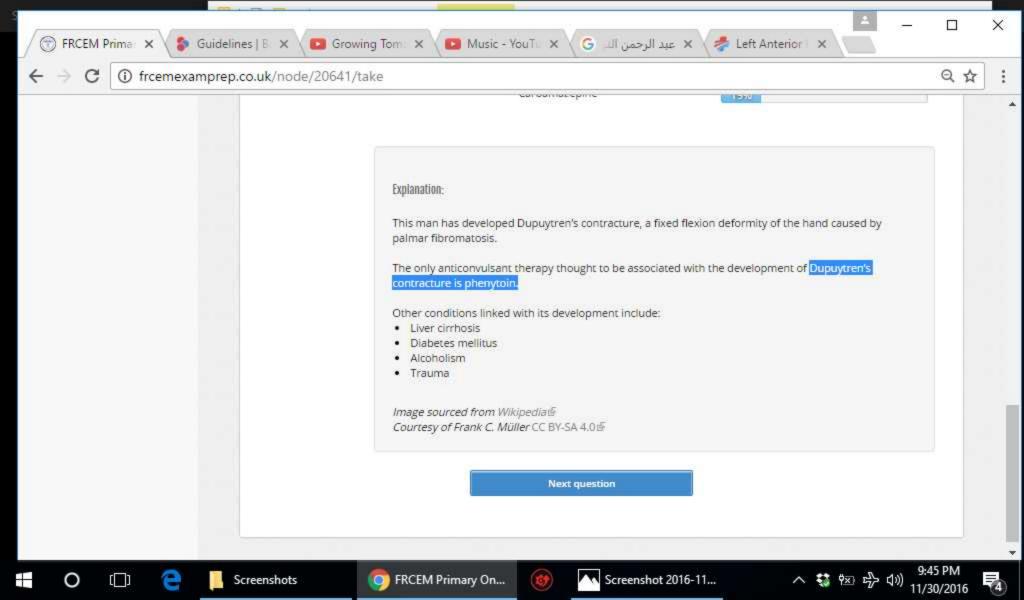
The adult oral dose is 100-200 mg 12-hourly. Intravenously it may be administered as a bolus dose of 2 mg/kg over 10 minutes followed b an infusion of 1.5 mg/kg/hour for one hour, reducing to 0.25 mg/kg/hour.

Flecainide should not be alone in the treatment of atrial flutter. If used alone there is a risk of inducing 1:1 atrioventricular conduction, with a consequent paradoxical increase in ventricular rate.

Flecainide is indicated only in patients without structural heart disease for the prevention, rapid control, or short-term prophylaxis of supraventricular and ventricular arrhythmias. The CAST trial showed a significant increase in sudden cardiac death and all-cause mortality in patients post-myocardial infarction, where is tended to be pro-arrhythmic, and in patients with an ejection fraction of < 40%. circ.ahajournals.org di

Recognized side effects of flecainide include:

- · Reversible liver toxicity
- Dizziness/vertigo
- · Nausea and vomiting
- Visual disturbance
- Parasthesiae
- · Interstitial lung disease



Adrenaline: Score 0 of 1

Which SINGLE statement regarding adrenaline is true?

Answer	Option	Question Statistics
×	The IV dose in adult cardiac arrest is 0.1 mg	1195
	It is not absorbed from the tracheal mucosa	296
	It is well absorbed orally	49.6
~	It is predominantly metabolized by COMT and MAO	70%
	It is a selective alpha-adrenergic receptor agonist	13 <sub>95</sub>

Tricyclic antidepressants (TCAs) are mainly used in the treatment of depression but are also used in the treatment of anxiety disorders, chronic pain conditions and attention-deficit hyperactivity disorder (ADHD). The majority of TCAs act primarily as serotonin-noradrenaline reuptake inhibitors (SNRIs) by blocking the serotonin transporter (SERT) and the noradrenaline transporter. This results in an elevation in the synaptic concentrations of serotonin and noradrenaline, and therefore an enhancement of neurotransmission. Many of the common side effects of TCAs are related to their antimuscarinic properties. These include: · Dry mouth and mucous membranes · Blurred vision Constipation · Urinary retention Cognitive impairment Other side effects include: Anxiety · Apathy and anhedonia Akathisia Confusion Sexual dysfunction Gynaecomastia and lactation Dysrrhythmias TCAs should not be used concomitantly with monoamine oxidase inhibitors (MAOIs), such as selegiline, and should be started at least 2 weeks after stopping the MAOI. There is a risk of developing serotonin toxicity is the two drug classes are used together. Serotonin syndrome may occur with TCA overdose, Features of this syndrome include CNS effects (including agitation and coma), autonomic instability (including hyperpyrexia) and neuromuscular excitability (including clonus and raised serum creatine kinase). Contraindications to the use of TCAs include: The recovery period from MI Heart block Arrhythmias Manic phase of bipolar affective disorder Acute porphyria

This patient has a classical presentation of temporal arteritis. Temporal arteritis, also known as giant cell arteritis (GCA), is a type of chronic vasculitis characterized by granulomatous inflammation in the walls of medium and large arteries. It usually affects people over 50 years of age.

### Clinical features include:

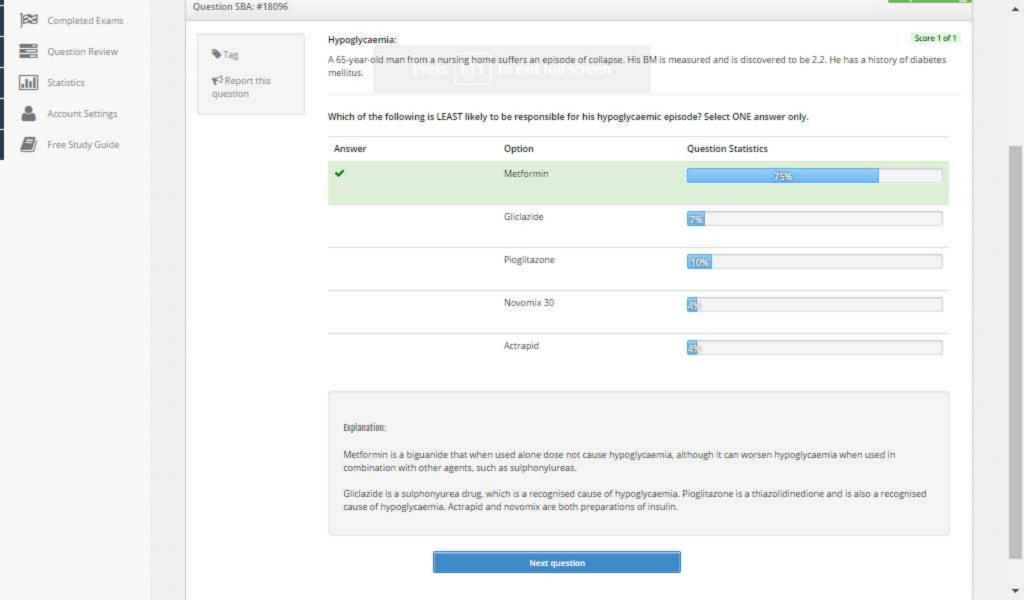
- Headache
- Scalp tenderness
- Jaw claudication
- Amaurosis fugax or sudden blindness (typically unilateral).

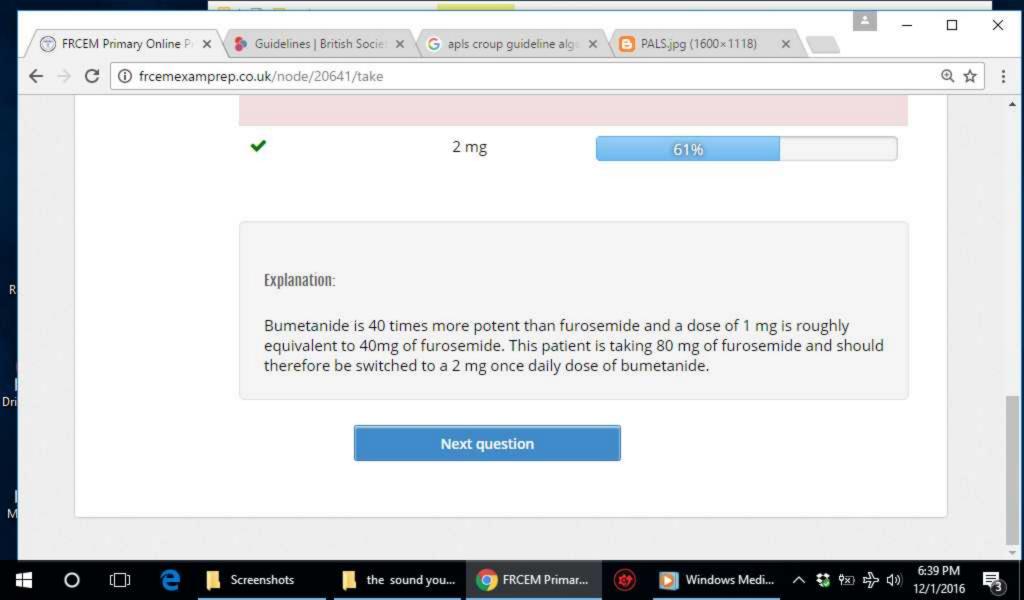
Some patients also present with systemic features such as fever, fatigue, anorexia, weight loss, and depression.

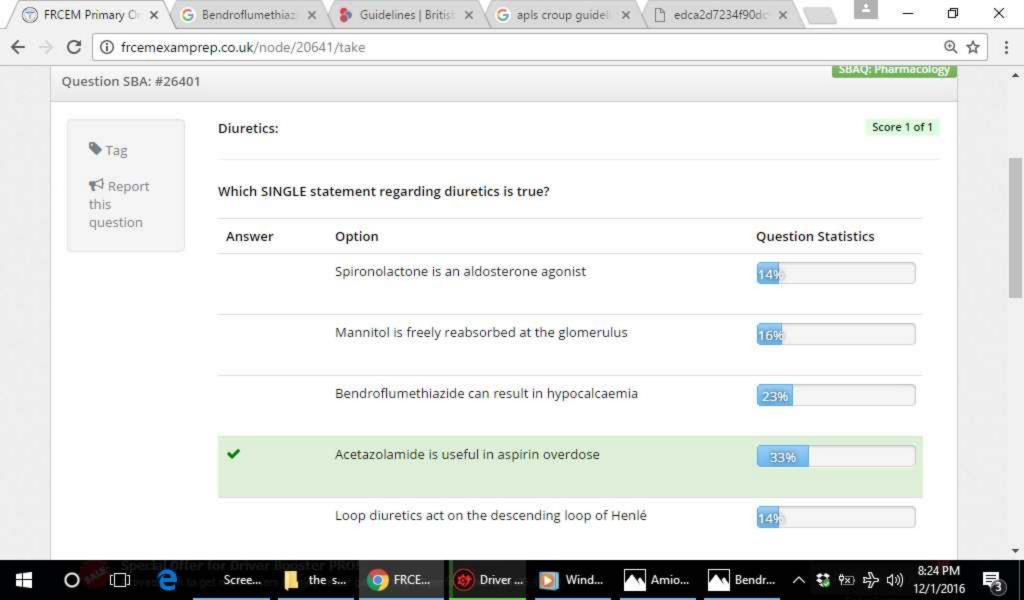
It is associated with polymyalgia rheumatica (PMR) in 50% of cases (bilateral upper arm stiffness, aching, and tenderness; pelvic girdle pain).

Visual loss occurs early in the course of disease and, once established, it rarely improves.

Early treatment with high-dose corticosteroids is imperative to prevent further visual loss and other ischaemic complications. If GCA is suspected high-dose glucocorticosteroid treatment should be initiated immediately (40 - 60 mg prednisolone daily). An urgent referral for specialist evaluation (same day ophthalmology assessment for those with visual symptoms) and temporal artery biopsy should also be organised.







The syndrome of inappropriate antidiuretic hormone (ADH) secretion (SIADH) is defined as the presence of hyponatremia and hypo-osmolality resulting from inappropriate, continued secretion or action of the hormone despite normal or increased plasma volume, which results in impaired water excretion.

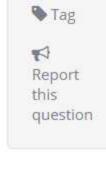
There are numerous causes of SIADH, of which carbamazepine is a well recognized example.

The causes of SIADH include:

- CNS damage: meningitis, subarachnoid haemorrhage
- Malignancy: small-cell lung cancer
- Drugs: carbamazepine, SSRIs, amitryptline, morphine
- · Infection: pneumonia, lung abscess, brain abscess
- · Endocrine: hypothyroidism

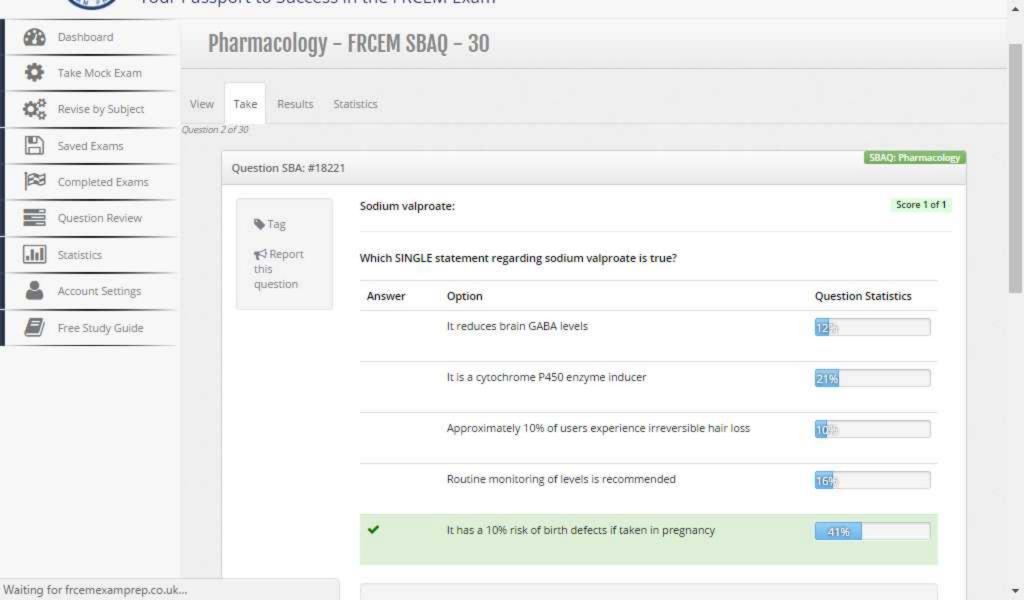
Demeclocycline is a tetracycline antibiotic that reduces the sensitivity of ADH receptors in the distal convoluted tubules. It is sometimes used in the management of SIADH that has responded to fluid restriction alone.

**Next question** 



Which of the following is LEAST likely to occur following the administration of thiopental sodium? Select ONE answer only.

Answer	Option	<b>Question Statistics</b>
	Decreased car <mark>d</mark> iac output	<b>6</b> %
	Hypotension	9%
*	Decreased vasopressin secretion	56%
	Respiratory depression	15%
	Decreased renal blood flow	13%



Sodium valproate is a medication primarily used in the treatment of epilepsy, but also used to manage migraine, chronic pain disorders, and bipolar affective disorder. It has a broad spectrum of anticonvulsant activity, but is primarily used in the treatment of tonic-clonic seizures, absence seizures, and myoclonic seizures. It is also used as a second-line treatment of partial seizures (including temporal lobe epilepsy) and infantile spasms.

Sodium valproate is thought to act via GABA-ergic inhibition. It is a weak inhibitor of sodium channels and is also a weak inhibitor of enzymes that deactivate GABA, such as GABA transaminase, it therefore increases brain GABA levels.

The adult oral dose is 600-2500 mg daily in two divided doses. The intravenous dose is 400-2500 mg daily in divided doses.

Sodium valproate is a cytochrome P450 enzyme inhibitor and therefore may potentiate the effects of other anti-epileptics, such as phenytoin.

The routine monitoring of sodium valproate levels is not recommended but the checking of levels is advised in the case of side effects or overdose.

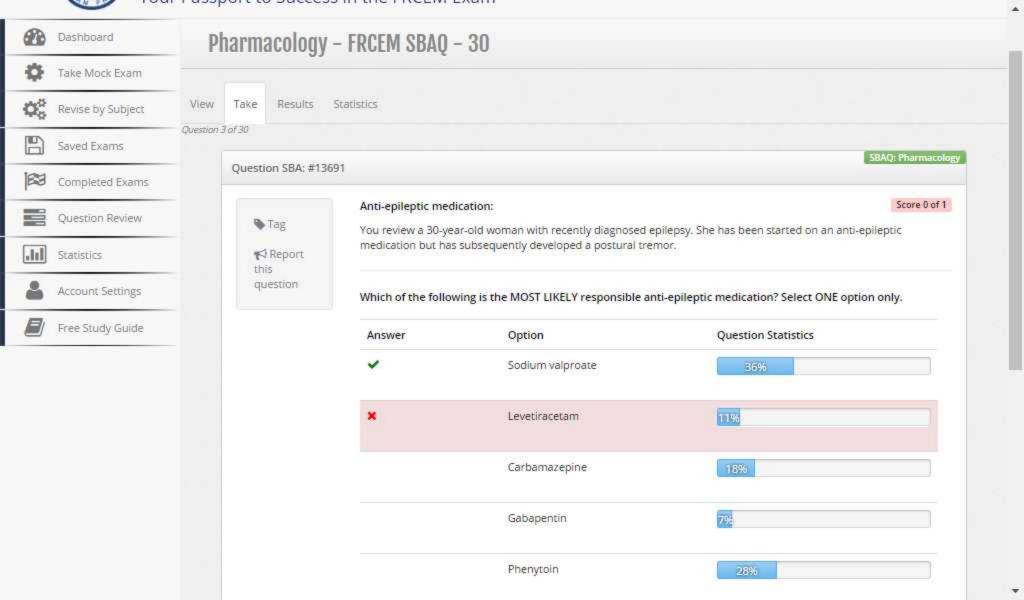
The effective plasma range is 40-100 mg/l.

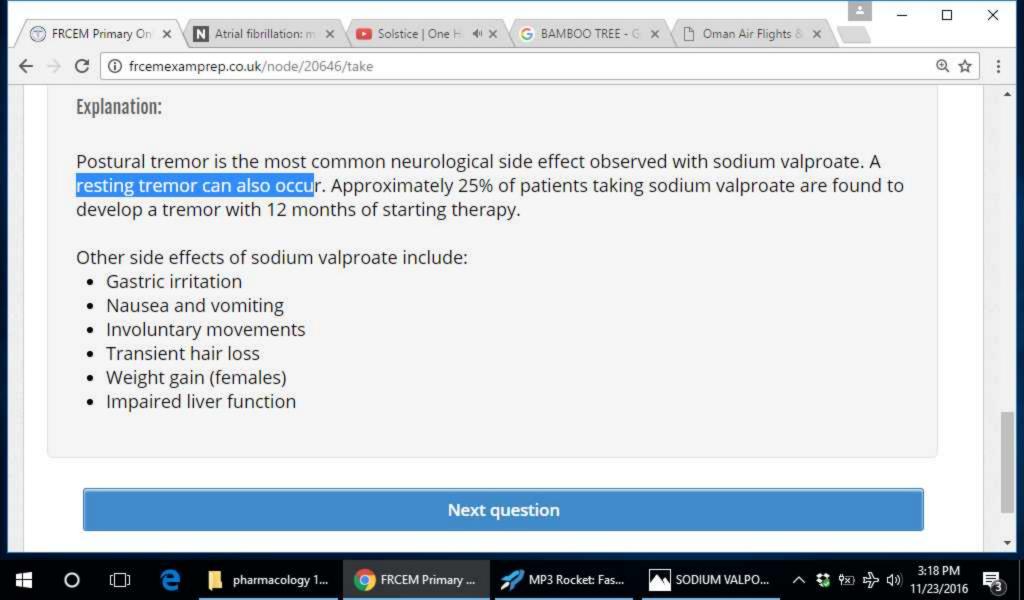
Approximately 10% of users experience hair loss, but this is reversible on discontinuation of the drug. The hair re-growth tends to be 'curly'.

Other side effects include:

- Thrombocytopaenia
- Ataxia
- Hepatitis
- Weight gain
- Amenorrhoea
- Gynaecomastia

The risk of birth defects in mothers taking sodium valproate is 2-5 times higher than with other frequently used antiepileptic drugs and it has an overall associated rate of birth defects of around 10%.





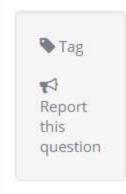
Although statins are for the most part relatively safe and well tolerated they are a well-recognized cause of myopathy and myotoxicity. Statins can cause a spectrum of myotoxicity varying from myalgia to rhabdomylosis in the most severe cases.

Rhabdomylosis is the most severe adverse effect of statins, and can result in renal failure, disseminated intravascular coagulation, and even death.

The range of myotoxicity associated with statins is as follows:

- Myalgia muscle symptoms without elevation of creatine kinase (CK)
- Asymptomatic mopathy elevated CK without muscle symptoms
- Myositis muscle symptoms with CK elevated < 10 x upper limit of normal</li>
- Rhabdomyolsis muscle symptoms, CK elevated > 10 x upper limit of normal with potential myoglobinuria and renal failure

Most statins are metabolized by the cytochrome P450 enzyme system and co-prescription with drugs that are potent inhibitors of cytochrome P450 can significantly increase the plasma concentration of the statin. This in turn increases the risk of myopathy. A commonly cited example of this is the use of the macrolide antibiotics erythromycin and clarithromycin, which when co-prescribed with statins are associated with an increased risk of myopathy, hospital admission with rhabdomylosis, acute kidney injury, and all cause mortality.



### Drug-interactions:

A 56-year-old man that is prescribed simvastatin for hypercholesterolaemia develops deep muscle pains following a course of antibiotics for a chest infection. He is subsequently admitted to hospital where he is found to be in acute renal failure and his creatine kinase (CK) level is 1260 units/litre.

Score 1 of 1

Which of the following antibiotics is he most likely to have been prescribed? Select ONE answer only.

Ontion

Quarties Statistics

Answer	Option	Question Statistics
	Amoxicillin	20%
	Co-amoxiclav	20/3
	Doxycycline	21%
	Cefalexin	<b>6</b> %
~	Clarithromycin	66%



## 7:57 PM

50%

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Which of the following is the 'intubating' dose of suxamethonium? Select ONE answer only.

Answer	Option	<b>Question Statistics</b>
	2-4 µg/kg	11%
	0.5-2 μg/kg	13%
	2-4 mg/kg	16%
	4-6 µg/kg	5%
~	0.5-2 mg/kg	54%

## **Explanation:**

The 'intubating' dose of suxamethonium is 0.5-2 mg/kg and the usual single dose for an adult is between 50-100 mg intravenously. Infants and younger children are relatively resistant to suxamethonium and usually require a dose of 1-2 mg/kg.

potential is prevented. The dose of suxamethonium is 0.5-2 mg/kg and the usual single dose for an adult is between 50-100 mg intravenously. Infants and younger children are relatively resistant to Severe muscle trauma Hyperkalaemia · History of malignant hyperthermia Recognized adverse effects of suxamethonium include: Bradycardia Hyperkalaemia Raised intracranial pressure · Raised intraocular pressure Prolonged paralysis Anaphylaxis · Malignant hyperthermia

Explanation:

relaxation and short-term paralysis, usually to facilitate endotracheal intubation.

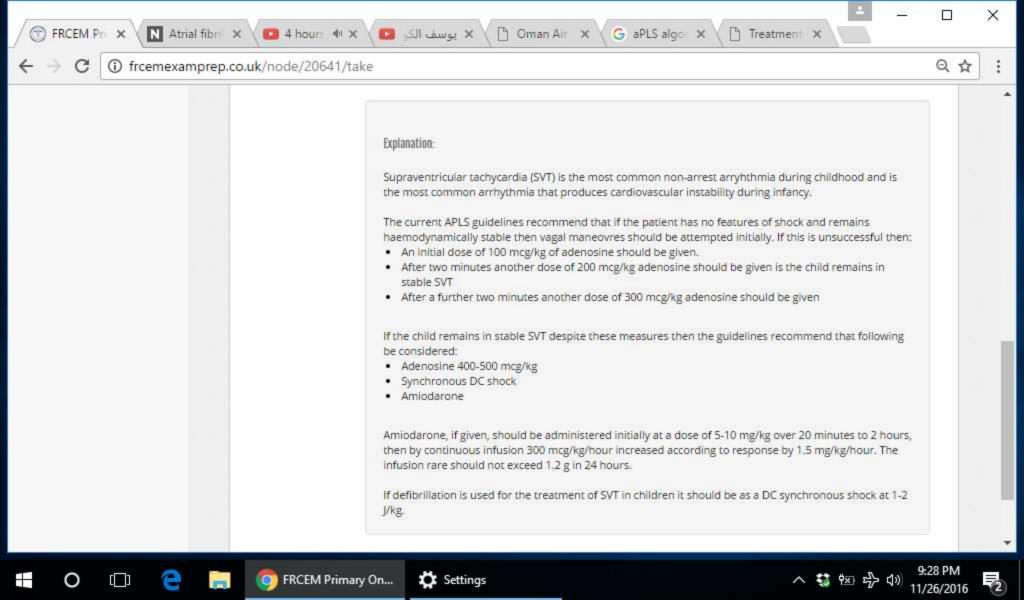
Suxamethonium is a depolarizing neuromuscular blocker that is used to induce muscle

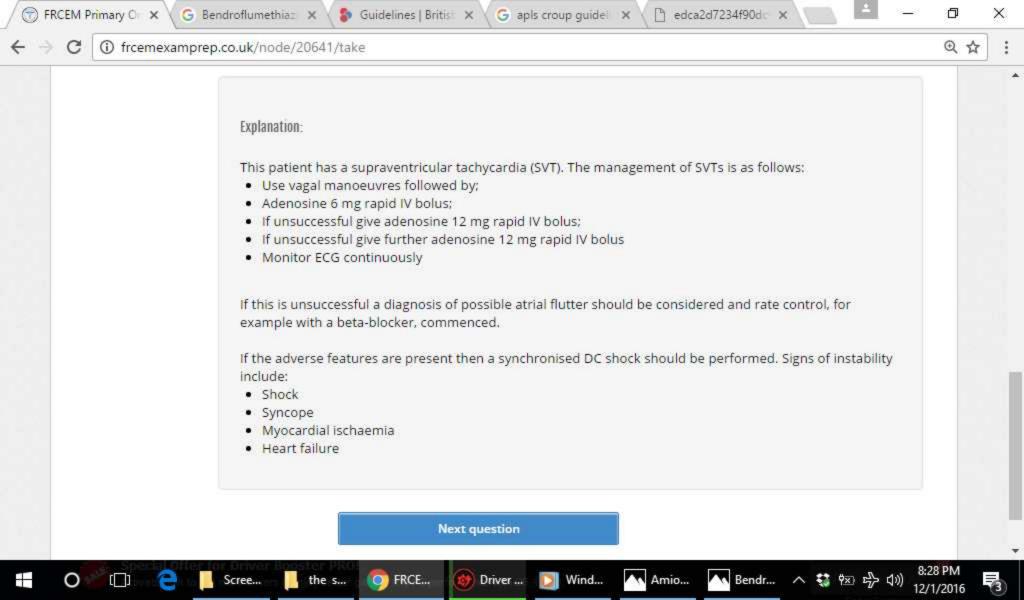
Suxamethonium causes a 'persistent' depolarization by mimicking the effects of acetylcholine without being rapidly hydrolysed by acetylcholinesterase. It therefore inhibits the action of acetycholine at the neuromuscular junction and the propagation of an action

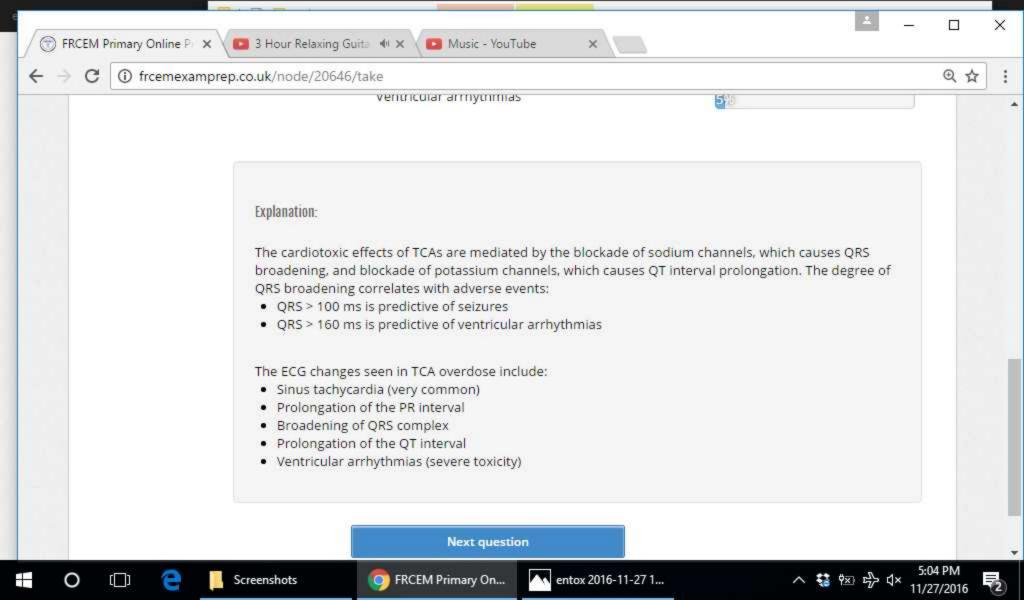
suxamethonium and usually require a dose of 1-2 mg/kg. The onset of action occurs within 30 seconds and the duration of action is 3-5 minutes. Suxamethonium is contraindicated in patients with recent burns but can be given in the

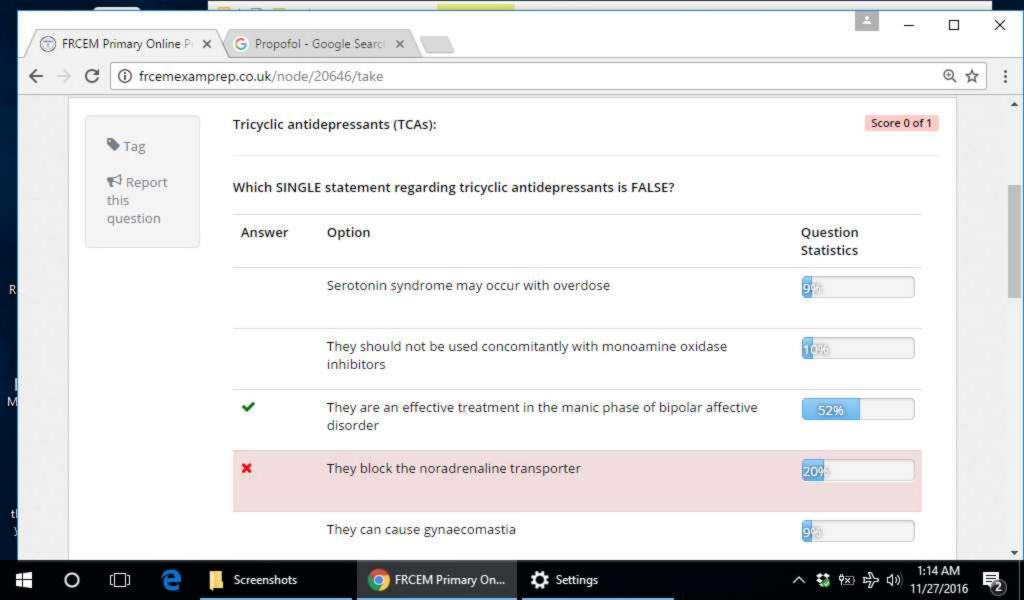
first 24 hours following the burn. It is also contraindicated in patients with spinal cord trauma causing paraplegia, it can be given immediately after the injury but should be avoided from approximately day 10 to day 100 after the injury.

Other contraindications to the use of suxamethonium include:









Tricyclic antidepressants (TCAs) are mainly used in the treatment of depression but are also used in the treatment of anxiety disorders, chronic pain conditions and attention-deficit hyperactivity disorder (ADHD).

The majority of TCAs act primarily as serotonin-noradrenaline reuptake inhibitors (SNRIs) by blocking the serotonin transporter (SERT) and the noradrenaline transporter. This results in an elevation in the synaptic concentrations of serotonin and noradrenaline, and therefore an enhancement of neurotransmission.

Many of the common side effects of TCAs are related to their antimuscarinic properties. These include:

- · Dry mouth and mucous membranes
- Blurred vision
- Constipation
- Urinary retention
- Cognitive impairment

#### Other side effects include:

- Anxiety
- · Apathy and anhedonia
- Akathisia
- Confusion
- Sexual dysfunction
- · Gynaecomastia and lactation
- Dysrhythmias

Dysrhythmias

TCAs should not be used concomitantly with monoamine oxidase inhibitors (MAOIs), such as selegiline, and should be started at least 2 weeks after stopping the MAOI. There is a risk of developing serotonin toxicity is the two drug classes are used together.

Serotonin syndrome may occur with TCA overdose. Features of this syndrome include CNS effects (including agitation and coma), autonomic instability (including hyperpyrexia) and neuromuscular excitability (including clonus and raised serum creatine kinase).

Contraindications to the use of TCAs include:

- The recovery period from MI
- · Heart block
- Arrhythmias
- · Manic phase of bipolar affective disorder
- Acute porphyria

Report this question

### Tetanus prophylaxis:

Score 1 of 1

The following patients have suffered wounds or injuries and have been managed as described in a local walk in centre.

### Which of the following patients has received appropriate tetanus cover? Select ONE answer only.

Answer	Option	Question Statistics
	25-year-old from Romania, cut hand on a drinking glass, uncertain of vaccination history - receives immunoglobulin and vaccination	45
	8-year-old UK resident, cut hand on a kitchen knife, had primary course of vaccinations as a baby plus booster at 4 years - receives vaccination	<b>5</b> 000
•	80-year-old diabetic, a rusty nail went through his shoe and into his foot when walking in the woods, uncertain of vaccination history - receives vaccination and immunoglobulin	67%
	30-year-old, electrical burn to torso with large amount of devitalised tissue - has had all vaccinations so no need for vaccination or immunoglobulin	Rio .
	28-year-old man from Poland, cuts leg whilst working in garden, wound heavily contaminated with soil, uncertain of vaccination history – receives vaccination	17,000

# 25 year old from Romania, cut hand on a drinking glass, uncertain of vaccination history - receives immunoglobulin and vaccination:

This is not a tetanus prone wound and therefore immunoglobulin is not indicated. As you cannot be sure of his vaccination history the best practice would be to take opportunity to commence course of tetanus vaccinations for cover later in life. The first vaccination should be given at the time of presentation, the patient's own GP should then check vaccination history and arrange the remainder of course as indicated.

# 8-year-old UK resident, cut hand on a kitchen knife, had primary course of vaccinations as a baby plus booster at 4 years - receives vaccination:

This child's tetanus vaccinations are on schedule and the next booster should not to be given early. This is not a tetanus prone wound so immunoglobulin is not indicated.

## 80-year-old diabetic, a rusty nail went through his shoe and into his foot when walking in the woods, uncertain of vaccination history - receives vaccination and immunoglobulin:

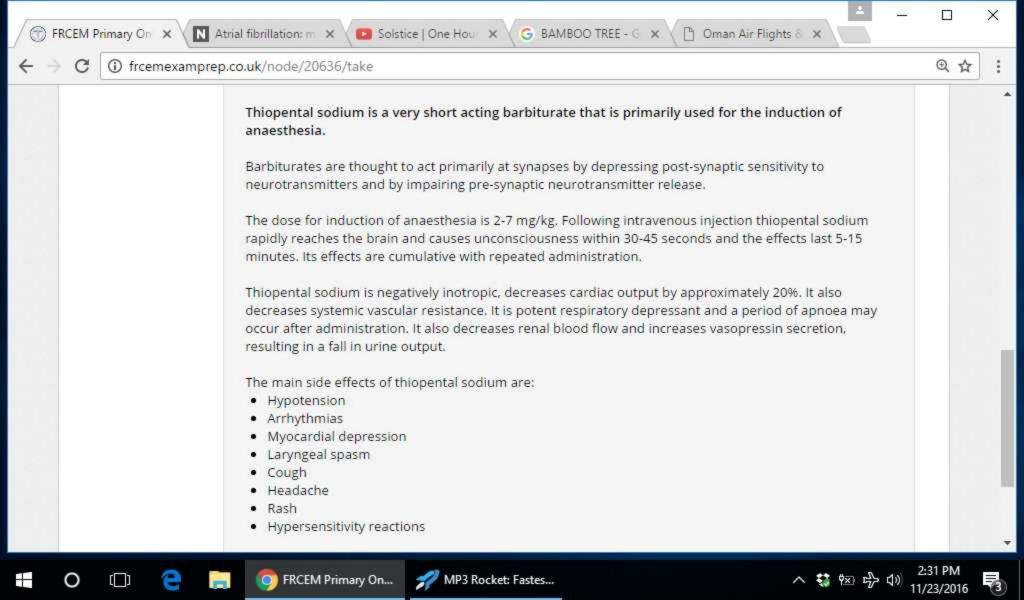
An 80-year-old UK resident may not have received a course of tetanus vaccinations (the vaccination was introduced in 1961). The vaccination should be given in the walk in centre and the patient's own GP contacted to confirm vaccination history and arrange the remainder of course as indicated. This is a tetanus prone wound (puncture wound and potential contact with soil), therefore in a patient with incomplete tetanus vaccinations, immunoglobulin would be indicated.

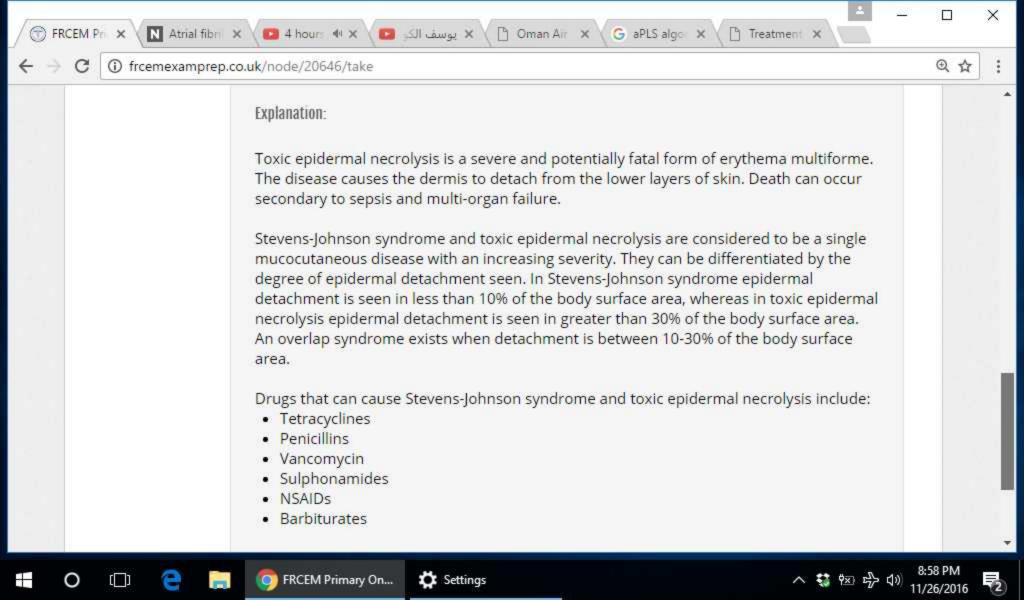
## 28-year-old man from Poland, cuts leg whilst working in garden, wound heavily contaminated with soil, uncertain of vaccination history – receives vaccination:

This is a tetanus prone wound in a patient with an uncertain vaccination history. The best course of action in this case would therefore be give both the vaccination and immunoglobulin at the walk in centre and then contact the patient's own GP to check vaccination history and arrange the remainder of course as indicated.

## 30-year-old, electrical burn to torso with large amount of devitalised tissue - has had all vaccinations so no need for vaccination or immunoglobulin

This is a high-risk tetanus prone wound (large amount of devitalised tissue), therefore even if the patient has had a full course of vaccinations in the past, the guidelines recommend immunoglobulin. No further vaccination is required.

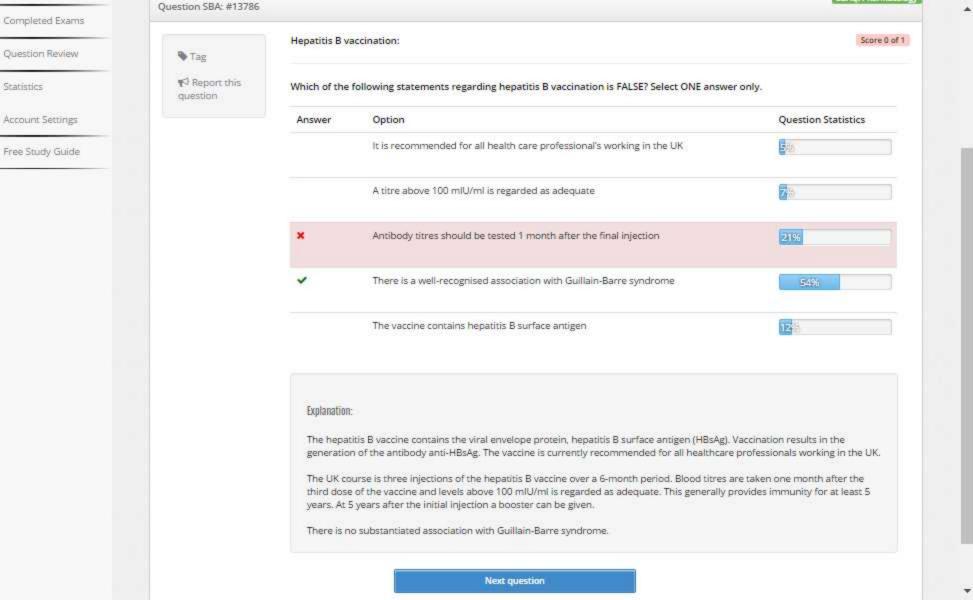




**Trimethoprim binds to dihydrofolate reductase** and inhibits the reduction of dihydrofolic acid (DHF) to tetrahydrofolic acid (THF). THF is an essential precursor in the thymidine synthesis pathway and interference with this pathway inhibits bacterial protein synthesis.

An overview of the different mechanisms of action of the various types of antimicrobial agents is shown below:

Mechanism of action	Examples	
	Penici <mark>lli</mark> ns	
Inhibition of cell wall synthesis	Cephalosporins	
	Vancomycin	
	Polymyxins	
Disruption of cell membrane function	Nystatin	
21-	Amphotericin B	
	Macrolides	
Inhihitian of protein conthocia	Aminoglycosides	
Inhibition of protein synthesis	Tetracyclines	
	Chloramphenicol	
	Quinolones	
habibitan af madain naid a matharia	Trimethoprim	
Inhibition of nucleic acid synthesis	5-nitroimidazoles	
	Rifampicin	
Anti-matabalia artivity	Sulfonamides	
Anti-metabo <mark>l</mark> ic activity	Isoniazid	



.iti

Vancomycin is a bactericidal antibiotic that acts by inhibiting cell wall synthesis in Grampositive bacteria. It prevents N-acetylmuramic acid (NAM)- and N-acetylglucosamine (NAG)-peptide subunits from being incorporated into the peptidoglycan matrix; which forms the major structural component of Gram-positive cell walls. The large hydrophilic molecule is able to form hydrogen bond interactions with the terminal D-alanyl-D-alanine moieties of the NAM/NAG-peptides. This binding of vancomycin to the D-Ala-D-Ala prevents the incorporation of the NAM/NAG-peptide subunits into the peptidoglycan matrix.

Due to the different mechanisms by which Gram-negative bacteria produce their cell walls and the various factors related to entering the outer membrane of Gram-negative bacteria, it is not active against Gram-negative bacteria.

Vancomycin is not absorbed orally and is excreted unchanged renally. It has a biological half-life in adults of 4 to 11 hours in an adult with normal renal function but this can increase to as long as 10 days in patients with impaired renal function.

It is important for the treatment of patients with septicaemia or endocarditis caused by methicillin-resistant strains of *Staphlyococcus aureus*. It can also be given orally for the treatment of antibiotic-associated pseudomembranous colitis (*C.difficile* infection)

#### Common side effects of vancomycin include:

- · Localised pain at injection site
- Thrombophlebitis

#### Rare side effects of vancomycin include:

- Renal failure (nephrotoxicity)
- Hearing loss (ototoxicity)
- · Toxic epridermal necrolysis
- Erythema multiforme
- Red man syndrome
- Blood dyscrasias

#### 

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Verapamil is a calcium channel-blocker used in the treatment of hypertension, angina, cardiac arrhythmias and most recently, cluster headaches.

Verapamil acts by blocking L-type calcium channels and has particularly powerful effects on the atrioventricular node (AV node), where conduction is entirely dependent on calcium spikes. It also inhibits the influx of Ca<sup>2+</sup> during the plateau phase of the action potential and therefore has a negatively inotropic effect.

The adult oral dose of verapamil is 240-480 mg in 2-3 divided doses. The corresponding intravenous (IV) dose is 5-10 mg administered over 30 seconds. The peak effect after IV injection occurs at 3-5 minutes and the duration of action is 10-20 minutes.

Verapamil has largely been replaced by adenosine in the treatment of acute supraventricular tachycardia (SVT) because adenosine is relatively safer, although oral verapamil is still used in the prophylaxis of SVT.

Verapamil should not be used in combination with beta-blockers or quinidine because the cumulative negatively inotropic effects are potentially catastrophic.

The side effects of verapamil include:

- Dizziness
- Flushing
- Nausea and vomiting
- 1<sup>st</sup> and 2<sup>nd</sup> degree heart block
- Precipitation of heart failure in patients with

●●●○○ OMANTEL 4G

1:14 AM

⊕ 76% ■

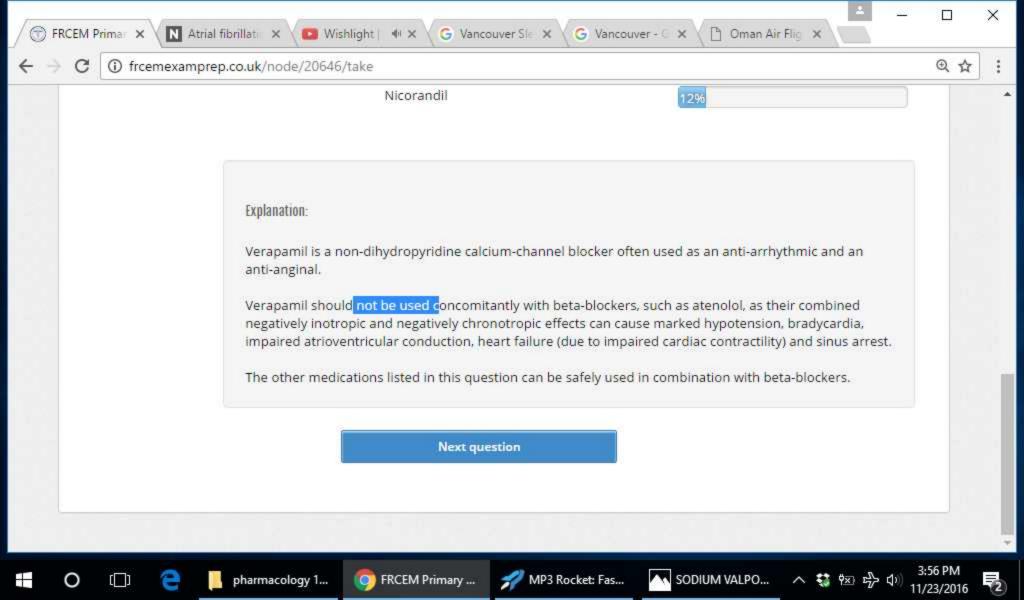
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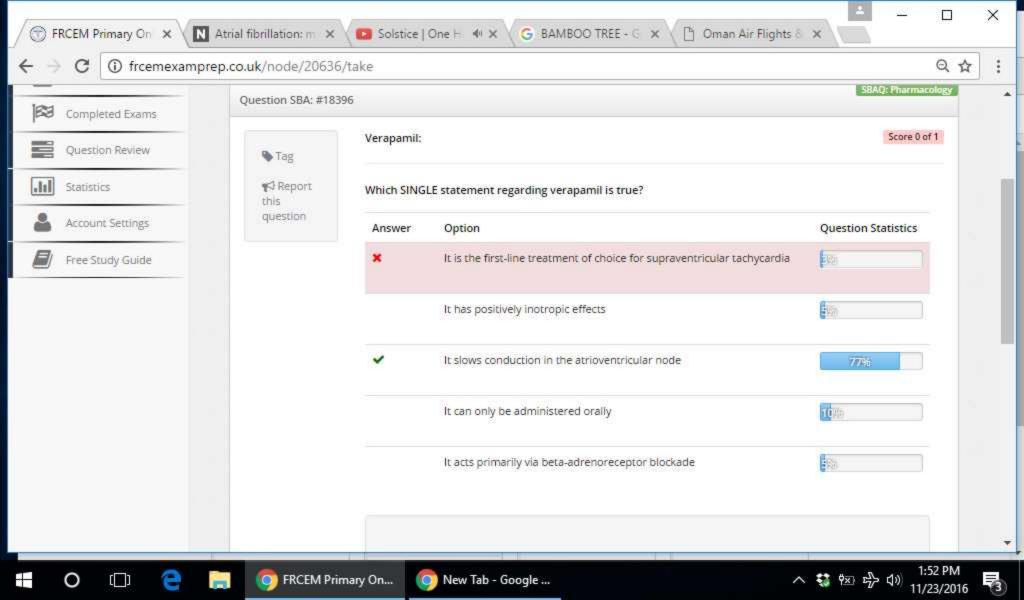
Verapamil should not be co-prescribed with which of the following drugs? Select ONE answer only.

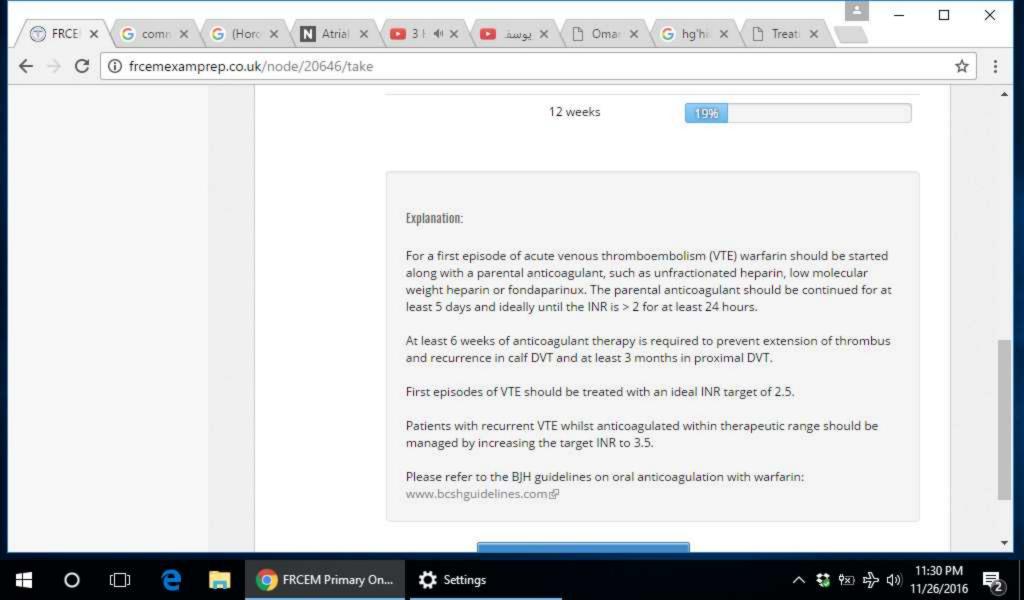
Answer	Option	<b>Question Statistics</b>
×	Warfarin	9%
~	Bisoprolol	81%
	Paracetamol	2%
	Simvastatin	6%
	Amoxicillin	2%

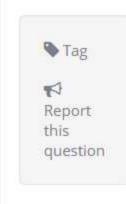
## **Explanation:**

Verapamil should not be used in combination with beta-blockers or quinidine because the cumulative negatively inotropic effects are potentially catastrophic.









Whooping cough:

Score 1 of 1

A 6-year-old boy is diagnosed as having whooping cough. There are two members of the household that are considered to be within a 'priority group' for post-exposure chemoprophylaxis.

Which of the following is the MOST appropriate antibiotic to be prescribed for this purpose? Select ONE answer only.

Answer	Option	Question Statistics
	Penicillin V	12%
	Co-amoxiclav	596
	Ciprofloxacin	9%
~	Erythromycin	58%
	Rifampicin	16%